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Podrid's Real-World ECGs

A Master's Approach to the Art and Practice of Clinical ECG Interpretation

Volume 6 Paced Rhythms, Congenital Abnormalities, Electrolyte Disturbances, and More

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Rahul Kakkar, MD · Peter A. Noseworthy, MD

Forewords by: Hein J.J. Wellens, MD · Roman W. DeSanctis, MD

cardiotext

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cardiotext PUBLISHING
Minneapolis, Minnesota

Cardiotext Publishing, LLC
3405 W. 44th Street
Minneapolis, Minnesota, 55410
USA
www.cardiotextpublishing.com

Additional information or any updates to this book may be found at:
http://cardiotextpublishing.com/podrid-ecg-interpretation-v6-paced-rhythms

Comments, inquiries, and requests for bulk sales can be directed to the publisher at:
info@cardiotextpublishing.com.

Cover design by Caitlin Altobell and Elizabeth Edwards;
interior design by Elizabeth Edwards

Library of Congress Control Number: 2016939559

ISBN: 978-1-935395-06-5
eISBN: 978-1-942909-04-0

These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy, daughter Sapna, and son Sanjay, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack.

Peter A. Noseworthy

Podrid's Real-World ECGs—The Complete Series

Podrid's Real-World ECGs: A Master's Approach to the Art and Practice of Clinical ECG Interpretation

Volume 1 The Basics

Volume 2 Myocardial Abnormalities

Volume 3 Conduction Abnormalities

Volume 4 Arrhythmias
- Part A: Core Cases
- Part B: Practice Cases

Volume 5 Narrow and Wide Complex Tachyarrhythmias and Aberration
- Part A: Core Cases
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Volume 6 Paced Rhythms, Congenital Abnormalities, Electrolyte Disturbances, and More

For more information about the other volumes in the series, please visit cardiotextpublishing.com

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Foreword

The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven's landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiodiagnostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today's cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled "Real-World ECGs," by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient's clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.

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These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important component for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, cardiotextpublishing.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

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Foreword

The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century, when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable "workhorse" in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of "Real-World ECGs" that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician's office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of *Podrid's Real-World ECGs* aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in *Podrid's Real-World ECGs* teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

This volume, the sixth in the series, delves into miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions. The other volumes focus on the

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basic approaches to reading any ECG as well as on other disease entities for which the ECG is useful:

- Essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the interpretation of all ECGs (Volume 1)
- Atrial and ventricular hypertrophy, acute myocardial ischemia, acute and chronic myocardial infarction, and pericarditis (Volume 2)
- AV and intraventricular conduction disturbances and enhanced AV conduction (Volume 3)
- Rhythm analysis, covering sinus, atrial, junctional, and ventricular arrhythmias (Volume 4)
- Narrow and wide complex tachyarrhythmias and aberration (Volume 5)

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs. Every ECG presents a clinical scenario to further enhance the student's skills at ECG analysis. Each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: www.realworldECGs.com

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers optional access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q&A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented, and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

Philip Podrid, MD
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Rahul Kakkar, MD
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Introduction

Pacemakers, Recording Issues (Standardization, Speed, Artifact), Lead Switch Electrolytes, Drugs, Congenital Abnormalities, and Required Abnormalities

Pacemakers

Pacemaker leads can be inserted into either the right atrium or right ventricle; these are single-chamber pacemakers. Leads can be inserted into both the right atrium and ventricle, and these are dual-chamber pacemakers. Lastly, leads may be inserted into the right atrium, right ventricle and coronary sinus over the left ventricle; these are biventricular pacemakers used for left ventricular pacing or cardiac resynchronization therapy as treatment for heart failure.

When a ventricular lead is in the right ventricle, the paced QRS complex has a left bundle branch block morphology, as the impulse originates in the right ventricle and is conducted via the myocardium to the left ventricle. Hence there is a broad R wave in lead I that is a bipolar lead that looks at the impulse as it travels right to left (with a leftward direction producing a broad R wave in this lead) and leads V5–V6, and deep QS complex in lead V1 (reflecting the impulse going away from this lead in a right to left direction). However, a left bundle branch block may have a QS complex across the precordium (*ie*, in leads V5–V6). In addition, a right ventricular lead near the intraventricular septum may produce a broad R wave in lead V1, which is not typical for a left bundle branch block.

When there is a biventricular pacemaker present, the initial ventricular activation is from the left ventricle and then travels via the ventricular myocardium to the right, resembling a right bundle branch block. Hence lead I, which is a bipolar lead that looks at the impulse as it travels right to left and usually has an R wave, as the impulse normally goes from right to left, will have an initial Q wave or a QS complex as the impulse is directed from left to right. Also seen may be a broad R wave in lead V1 and a QS complex in leads V5–V6, although as indicated this may also be seen with right ventricular pacing. Hence lead I is the most important lead that is diagnostic of a biventricular pacemaker.

Pacemaker electrode leads may be either bipolar or unipolar. Bipolar leads have the two poles (positive and negative) at the tip of the electrode lead, close to each other. Hence the electrical pathway or circuit is narrow, and the amount of electric energy generated is small, resulting in low-amplitude pacing stimuli, artifacts, or spikes on the ECG. Unipolar leads have one pole at the tip of the electrode catheter while the other pole is at the insertion into the pacemaker generator. Hence there is a large circuit, and the electrical impulse is of high amplitude, producing tall pacing stimuli or artifacts on the surface ECG.

A pacemaker may function in an asynchronous mode, *ie*, it paces continuously regardless of the underlying rate or rhythm. Most often, pacemakers function in a demand mode, *ie*, they are activated whenever the intrinsic heart rate falls below a preset lower rate limit of the pacemaker.

In order to define the basic function of the pacemaker, a five-letter code has been designated for describing pacing characteristics. However, most commonly used is a three-letter code. When a code includes only three or four characters, it can be assumed that the positions not mentioned are "O" or absent.

Position I

The first position reflects the chamber(s) paced. "A" indicates the atrium, "V" indicates the ventricle, and "D" means dual-chamber (*ie*, both the atrium and the ventricle).

Position II

The second position refers to the chamber(s) sensed. When there is sensing, the pacemaker functions in a demand mode, *ie*, it paces only when the intrinsic heart rate falls below the lower limit of the pacemaker. The letters are the same as those for the first position: "A" for atrium, "V" for ventricle, "D" for dual. An additional option "O" indicates an absence of sensing, *ie*, the device will pace automatically at a specified rate, ignoring any intrinsic rhythm. This is asynchronous of fixed-rate pacing.

Position III

The third position refers to how the pacemaker responds to a sensed event.

- I indicates that a sensed event inhibits the output pulse, *ie*, demand mode.
- T indicates that an output pulse is triggered in response to a sensed event.
- D indicates that there are dual modes of response. This designation is restricted to dual-chamber systems.
  a. If there is no atrial activity sensed, there is an atrial output pulse. If there is no ventricular event sensed after a programmable delay (AV delay) there is a ventricular output pulse. This is termed AV sequential pacing.
  b. If there is no atrial activity sensed, there is an atrial output pulse. If there is a native ventricular event sensed within the programmable AV delay, then the ventricular output pulse is inhibited. The pacemaker is functioning as an atrial pacemaker.
  c. An event sensed in the atrium inhibits the atrial output. If there is no ventricular event sensed after a programmable AV delay, there is a ventricular output output pulse. This is termed atrial sensed (or P-wave synchronous) ventricular pacing.

  There is a programmable delay between the sensed atrial event and the triggered ventricular output (AV delay) to mimic the normal PR interval. If the ventricular lead senses a native ventricular signal during the programmed delay, it will inhibit the ventricular output.

- O indicates no response to sensed input; it is most commonly used in conjunction with an "O" in the second position. In this situation, the pacemaker is function in an asynchronous mode.

Position IV

The fourth position reflects rate modulation, also referred to as rate responsive or rate adaptive pacing.

- R in the fourth position indicates that the pacemaker has rate modulation and incorporates a sensor to adjust its programmed paced heart rate in response to patient activity.
- O indicates that rate modulation is either unavailable or disabled. "O" is often omitted from the fourth position (*ie*, DDD is the same as DDDO).

Position V

The fifth position specifies only the location or absence of multisite pacing, defined as stimulation sites in both atria, both ventricles, more than one stimulation site in any single chamber, or a combination of these.

This feature is uncommon, and the fifth position is usually omitted.

- O means no multisite pacing.
- A indicates multisite pacing in the atrium or atria.
- V indicates multisite pacing in the ventricle or ventricles.
- D indicates dual multisite pacing in both atrium and ventricle.

There are several problems that may occur with a pacemaker and the ECG is often the first indication of a pacemaker problem:

1. **Pacing stimuli are present, but there is loss of capture; *ie*, there is no P wave or QRS complex following the pacing stimulus. This may result from:**
   a. Failure to sense a native complex (P wave or QRS complex) followed by a pacing stimulus at a time when the myocardium is refractory to the pacing impulse and cannot be stimulated or captured
   b. Lead dislodgement or malposition
   c. Fibrosis or inflammation of the myocardium at the site of the pacemaker electrode
   d. Increase in capture threshold such that the pacemaker output inadequate for capture
   e. Lead failure
2. **Pacing stimuli are present, but there is failure to sense a native P wave or QRS complex; *ie*, a native P or QRS complex is followed by an atrial or ventricular pacing stimulus after a short interval. This may result from:**
   a. An atrial or ventricular complex of low amplitude below the sensing threshold, *ie*, inadequate signal
   b. Pacemaker sensitivity set too low for sensing native P wave or QRS complex
   c. Ectopic complex originating at a distance from the pacemaker lead
   d. Lead failure
   e. Environmental electrical fields or noise detection, such as a magnet that deactivates normal sensing

3. Pacing stimuli are absent, *ie*, in the absence of a native P wave or QRS complex, there is no pacing stimulus seen. This presents with the absence of pacing stimuli with pauses in the rhythm that are longer than the programmed base rate of the pacemaker. This may occur as a result of:

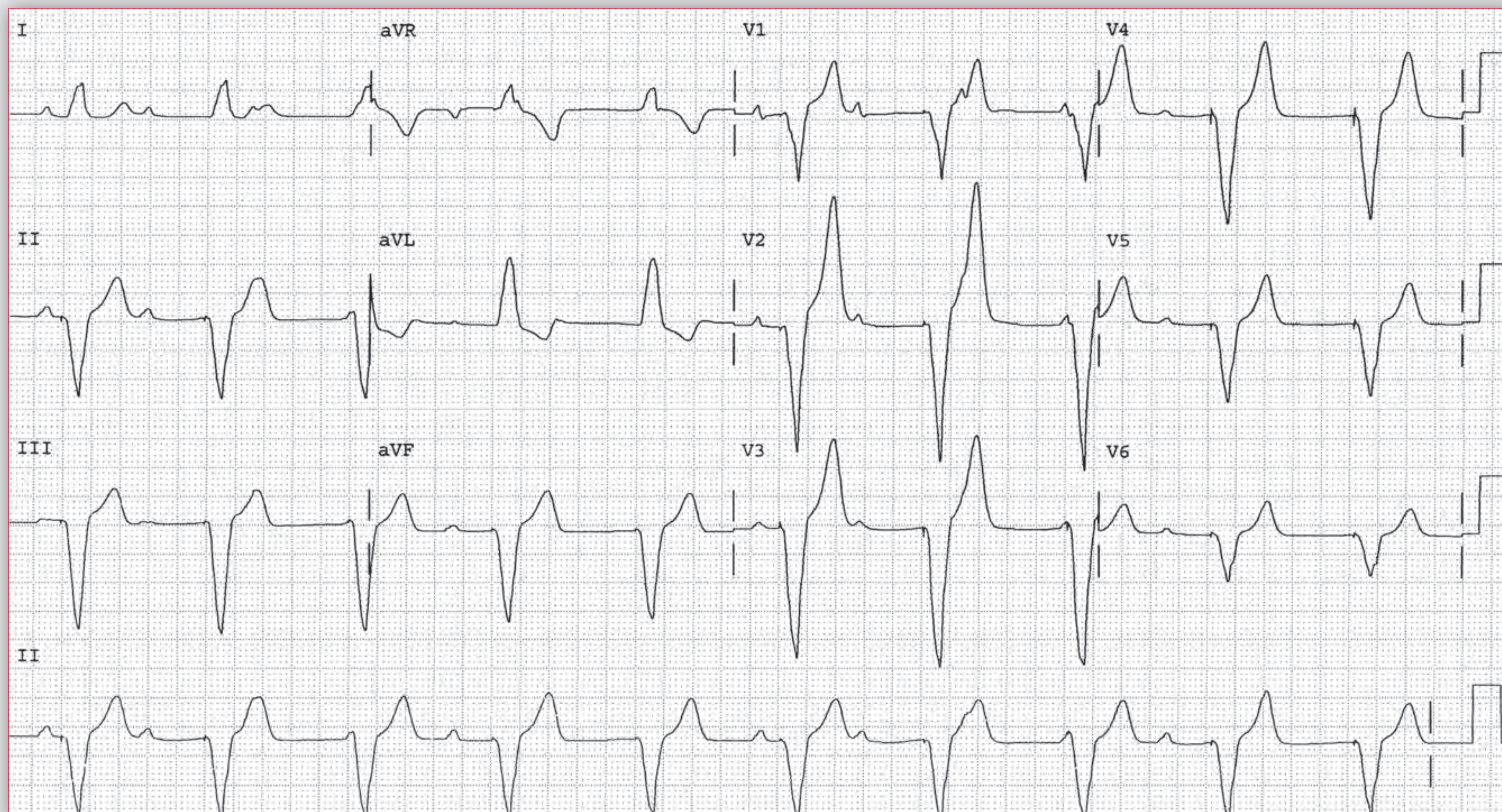
a. Oversensing an impulse that may not be seen on the surface ECG

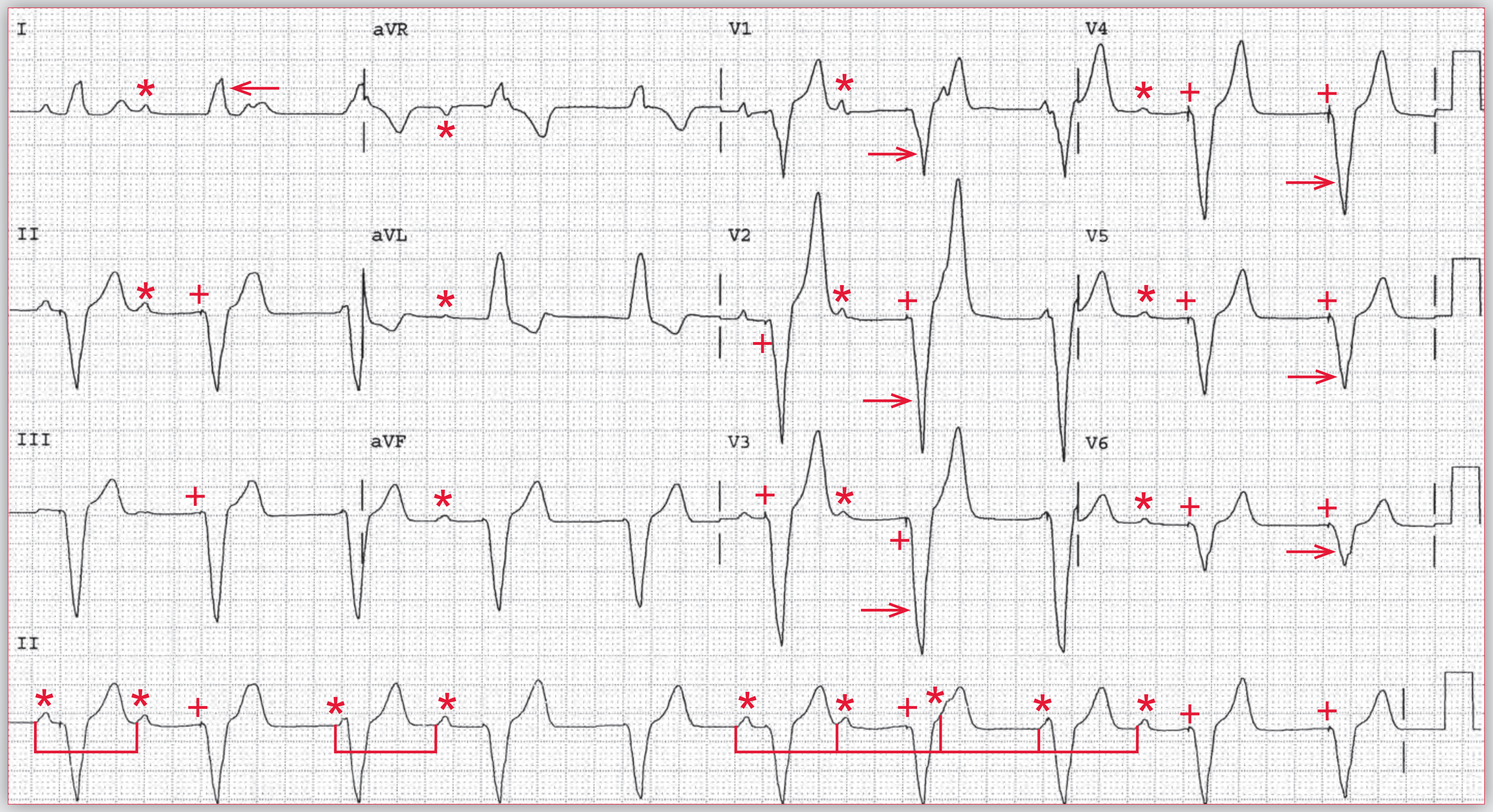
b. Electromagnetic interference sensed by the pacemaker as a native P wave or QRS complex, which then inactivates pacing output

c. Pulse generator problem, especially battery failure or battery end of life

d. Open circuit as may occur if lead fixation to the generator is inadequate, or if there is lead fracture ■

A 64-year-old man with aortic stenosis presents with intermittent lightheadedness and dyspnea. The following ECG is obtained while the patient is symptomatic.





ECG 1 Analysis: Sinus rhythm, AV dissociation with complete heart block, right ventricular pacing (VVI)

There is a regular rhythm with a rate of 60 bpm. There are P waves (*) seen at a regular rate of 86 bpm, and they are unrelated to the QRS complex, *ie*, they are dissociated. Although P waves are occasionally within T waves or the QRS, when they are seen they are on time (⊔). The P wave is positive in leads I, II, aVF, and V4–V6, and hence they are sinus P waves. The QRS complex duration is increased (0.20 sec) and it has a left bundle branch morphology with a broad R wave in lead I (←) and broad QS complexes in leads V1–V6 (→). The axis is extremely leftward between –30° and –90° (positive QRS complex in lead I and negative in leads II and aVF). The QT/QTc intervals are prolonged (480/480 msec) but are normal when the widened QRS complex duration is considered (380/380 msec). In some but not all leads, a pacing spike can be seen before each QRS complex (+), and this is right ventricular pacing (*ie*, it has a typical left bundle branch block pattern and especially a broad R wave in lead I). The pacing spikes are very small, often not obvious; hence a bipolar lead is present. The two electrodes or poles are close to each other at the tip of the pacing electrode. The circuit formed by these two close poles is small; hence the pacing spike has a low amplitude.

As there is no relationship between the sinus impulse and the ventricular paced beat, the pacemaker is functioning as a ventricular pacemaker and likely in a VVI mode, *ie*, it is a ventricular demand pacemaker. It paces the ventricle and senses ventricular impulses. If a native ventricular impulse is sensed, then the pacemaker is inhibited; if there is no ventricular impulse sensed, then the pacemaker delivers an output pulse. Since the atrial rate is faster than the pacing rate and there is no native QRS complex occurring in response to the P wave, there is underlying complete heart block.

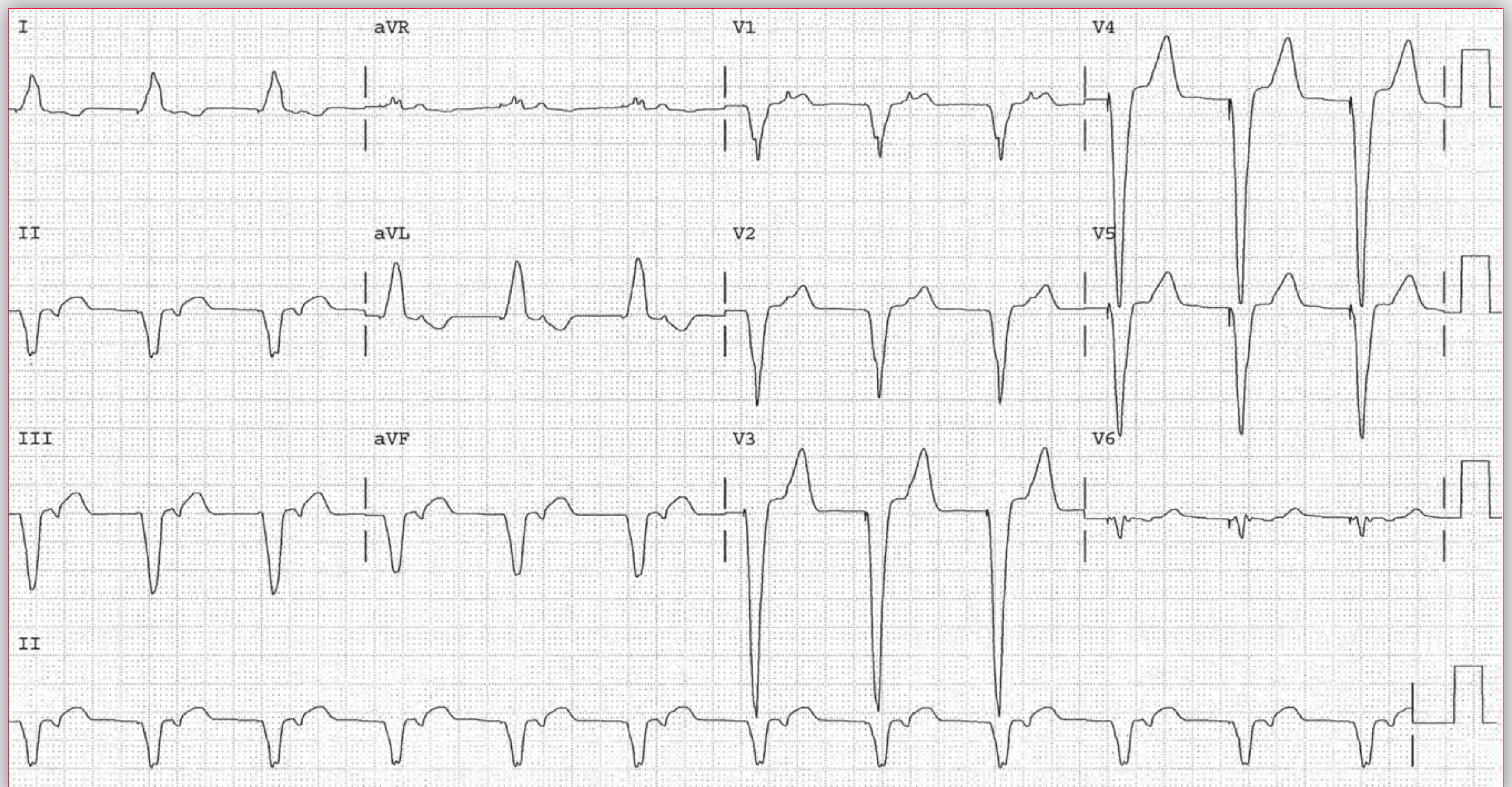
Loss of AV synchrony can result in significant symptoms, particularly in preload-dependent conditions such as aortic stenosis, hypertrophic cardiomyopathy, severe left ventricular dysfunction, or restrictive cardiomyopathy. In these situations, atrial contraction contributes up to 40% of stroke volume. As there is AV dissociation or loss of AV synchrony with VVI pacing when there is an underlying sinus, this will result in a reduction in stroke volume and hence symptoms. Such patients will do better with physiologic pacing, *ie*, atrial sensed ventricular pacing or P-wave synchronous ventricular pacing. ■

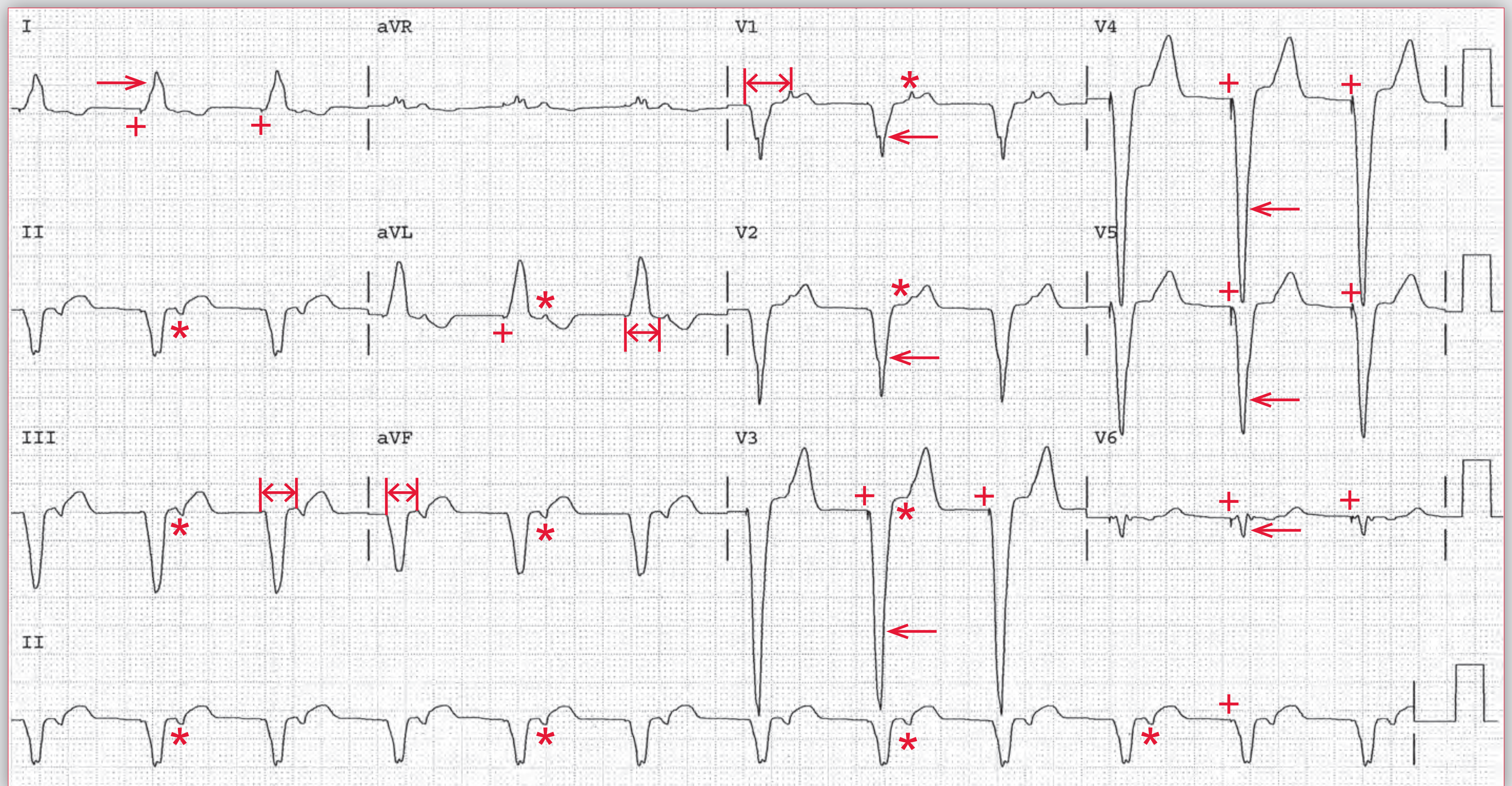
Notes

An asymptomatic patient has the following ECG. On physical examination there are some prominent waveforms noted in her neck.

What does the ECG show?

Should the patient be anticoagulated for atrial fibrillation?





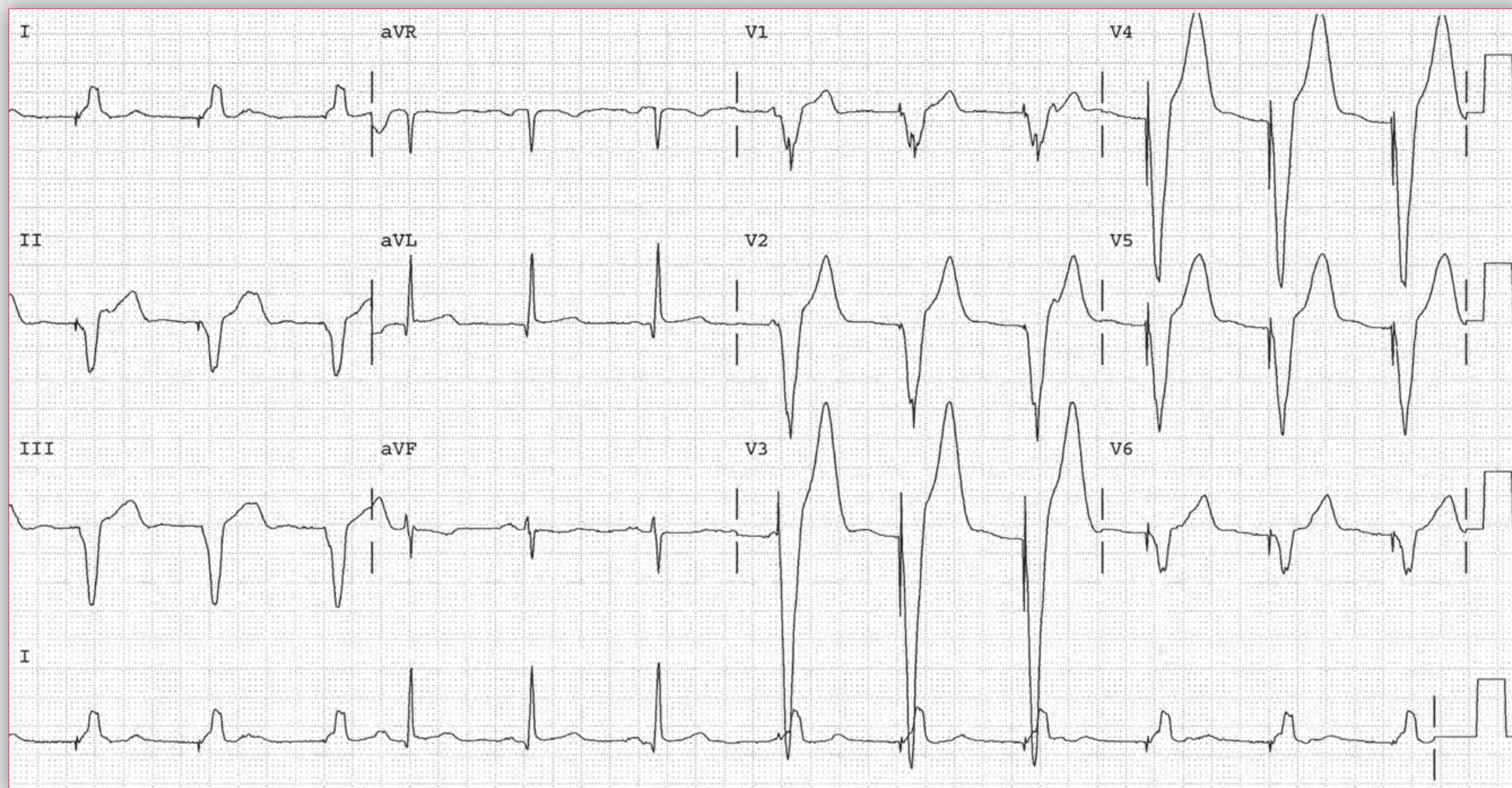
ECG 2 Analysis: Right ventricular pacing with retrograde atrial activation (V-A conduction)

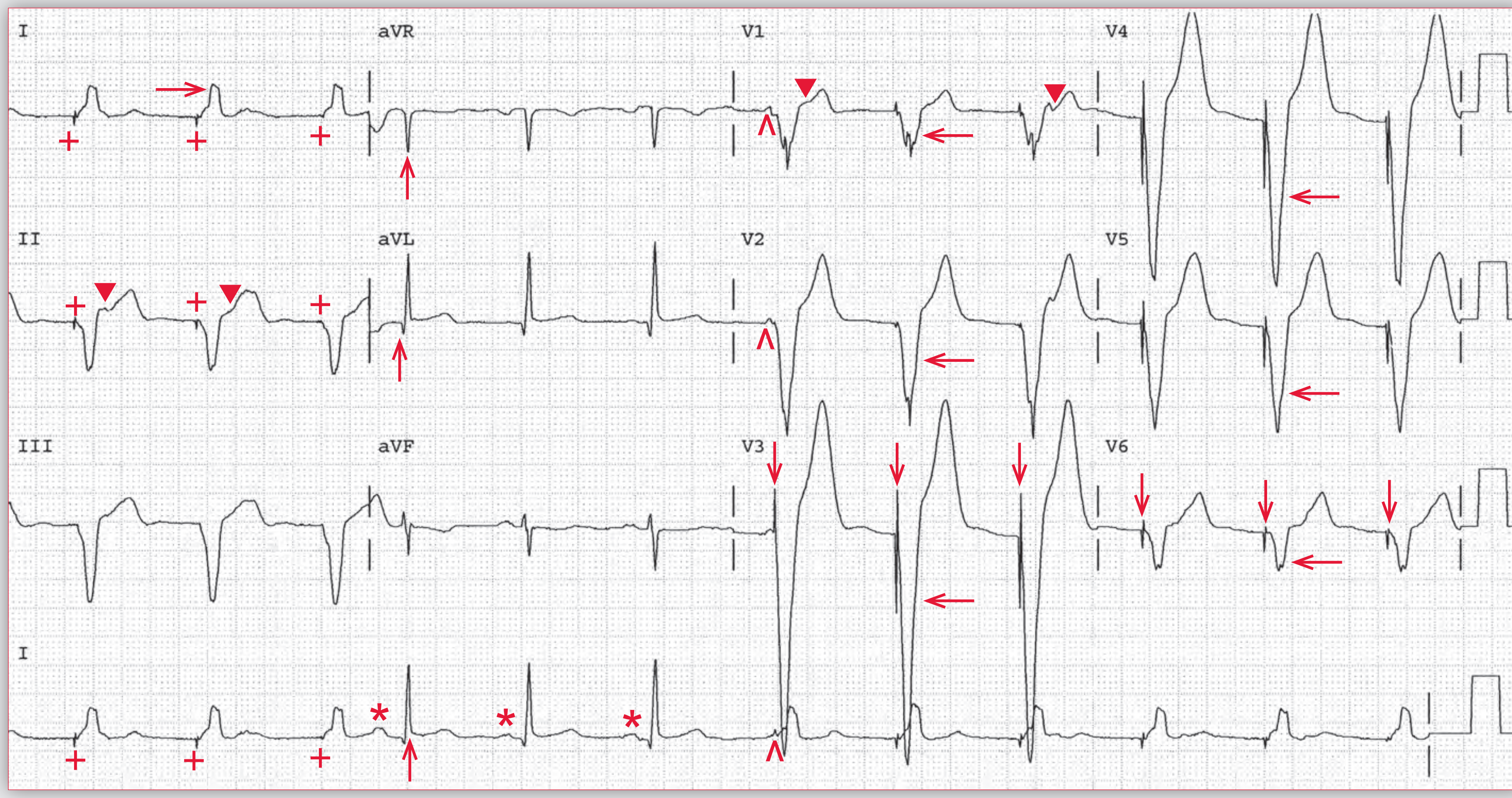
There is a regular rhythm at a rate of 70 bpm. The QRS complex duration is prolonged (0.18 sec) with a left bundle branch block morphology with a broad R wave in lead I (→) and a QS complex in leads V1–V6 (←). The QT/QTc intervals are prolonged (500/540 msec) but are normal when the prolonged QRS complex duration is considered (420/450 msec). A pacemaker output (+) (stimulus, artifact, or spike) is seen before each QRS complex in some but not all leads. Hence this is right ventricular pacing, likely VVI or demand ventricular pacing. Therefore, the extreme leftward axis (between –30° and –90°) is not the result of a left anterior fascicular block. There is a probably bipolar lead as the pacemaker stimulus is very small. A P wave (*) can be seen after each QRS complex, and there is a fixed relationship between the QRS complex and the P wave, *ie*, a fixed RP interval (↔). The P wave is negative in leads II, III, and aVF. Hence this is a retrograde P wave due to V-A conduction through the AV node. As there is a P wave seen, indicating atrial activation, the patient does not have atrial fibrillation and does not require anticoagulation. The prominent waveform in the neck is an "A wave" that has an increased amplitude as a result of atrial contraction against a closed tricuspid (and mitral) valve that occurs as a result of simultaneous atrial and ventricular contraction. ■

Notes

A 65-year-old asymptomatic woman comes for routine clinical evaluation. On physical examination, you notice intermittent strong pulsations of the neck veins that time with systole and a I/VI holosystolic murmur at the left sternal border that increases with inspiration. You obtain the following ECG.

What is the most likely etiology of the neck pulsations?





ECG 3 Analysis: Sinus rhythm with intermittent native ventricular conduction and right ventricular demand pacing (VVI) with AV dissociation

The first three QRS complexes are wide with a left bundle branch block morphology with a broad R wave in lead I (→) and a QS complex in leads V1–V6 (←), and there is a pacemaker impulse or spike before them (+). They are at a rate of 70 bpm. The QRS complex is wide (0.18 sec) and there is no obvious P wave before any of these complexes. Hence these complexes are the result of right ventricular pacing. The next three QRS complexes are narrow (0.08 sec) and there is a P wave (*) before each one with a fixed PR interval (0.18 sec). The P wave is positive in leads I and aVF and hence these are likely sinus P waves. The first of these narrow complexes (fourth QRS complex) is early (↑) as there is resumption of a sinus rhythm with a rate faster than the pacemaker. The rate of the sinus complexes is 72 bpm. As a result, there is no evidence of ventricular pacing, as the sinus rate is faster than the lower limit of the pacemaker rate (*ie*, 70 bpm), resulting in inhibition of the pacemaker output. Hence this is a demand ventricular pacemaker, *ie*, functioning in a VVI mode. The QT/QTc intervals of the sinus complexes are normal (360/395 msec).

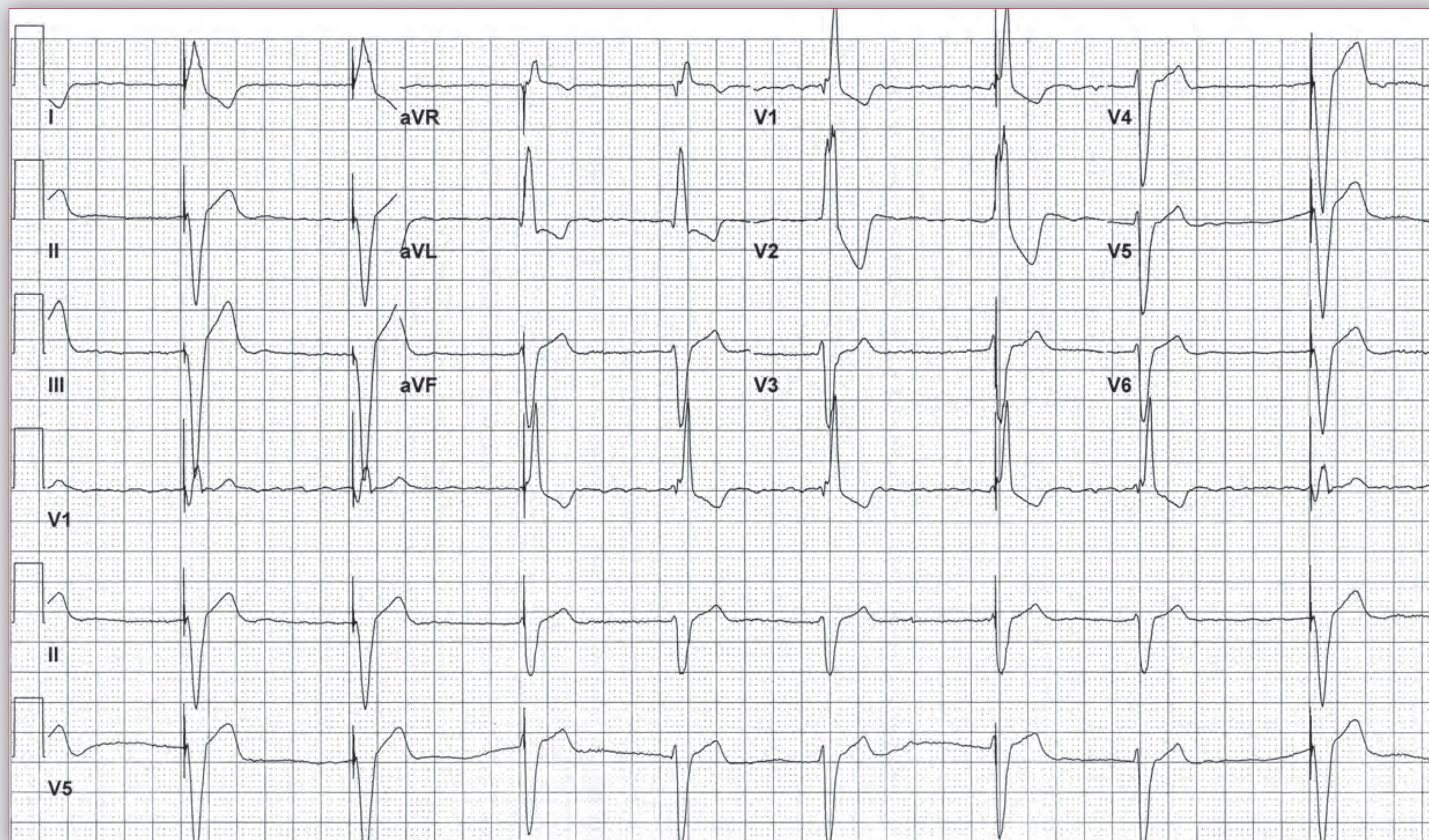
Following these three sinus complexes ventricular pacing resumes and pacemaker stimuli can be seen (↓). There is a P wave seen before the first of these paced complexes (^) (the seventh QRS complex) with a short PR interval. The P wave does not capture the ventricle (the PR interval is shorter than the baseline PR interval when there is sinus capture), but there is an appropriately timed paced complex. Since the sinus rate has slowed to below the lower rate limit of the pacemaker (*ie*, 70 bpm), the demand ventricular pacemaker resumes its normal function and paces. Although there are no obvious P waves seen during ventricular pacing, there are subtle changes in the T waves (▼), especially obvious in leads II and V1, which are superimposed P waves. They appear to be dissociated and at a rate of 62 bpm, which is below the lower rate limit of the pacemaker (*ie*, 70 bpm, which is the pacing rate).

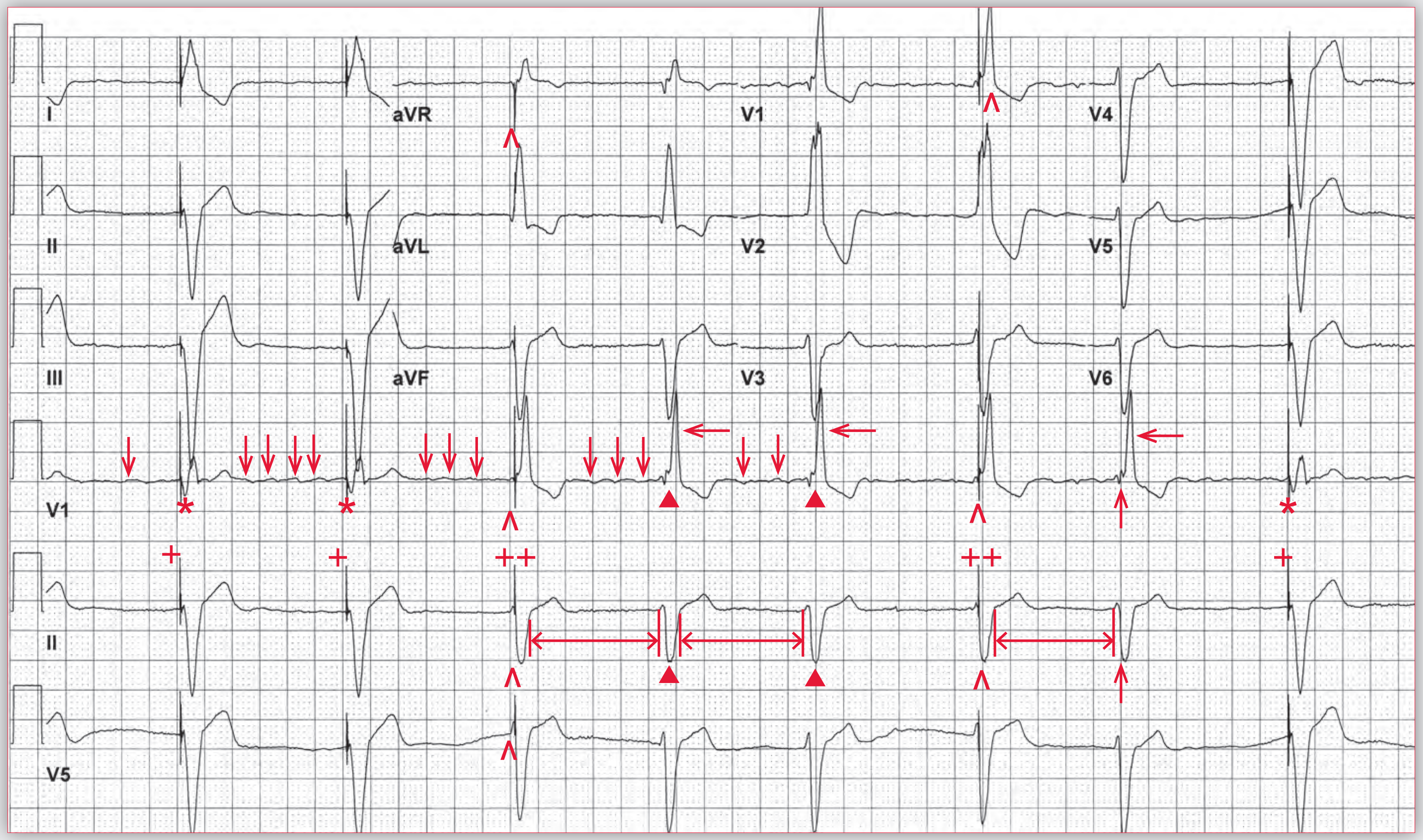
Jugular venous pulsations are biphasic and consist of two waves: (1) the A wave (atrial contraction at end-diastole) and (2) the V wave (venous return to the right atrium during systole). Tall V waves during systole can be the result of significant tricuspid regurgitation; however, these are typically observed with every beat and not intermittently. Additionally, the patient only has a I/VI murmur, suggestive of mild tricuspid regurgitation that is less likely to result in prominent V waves. However, intermittent strong jugular venous pulsations are more commonly due to “cannon A waves” from AV dissociation, when atrial contraction occurs against a closed tricuspid valve during ventricular systole. When there is VVI pacing, the P waves are dissociated from the ventricular complexes, being seen within the T waves, *ie*, there is AV dissociation and hence the presence of cannon A waves. ■

Notes

You are asked to evaluate a patient with pneumonia urgently in the emergency department for pacemaker malfunction or lead dislodgement. You are told that the pacemaker is pacing with varying QRS morphologies. The patient has been hemodynamically stable. The following ECG is obtained.

What is your diagnosis?





ECG 4 Analysis: Atrial fibrillation, intermittent demand, right ventricular pacing (VVI), pseudofusion

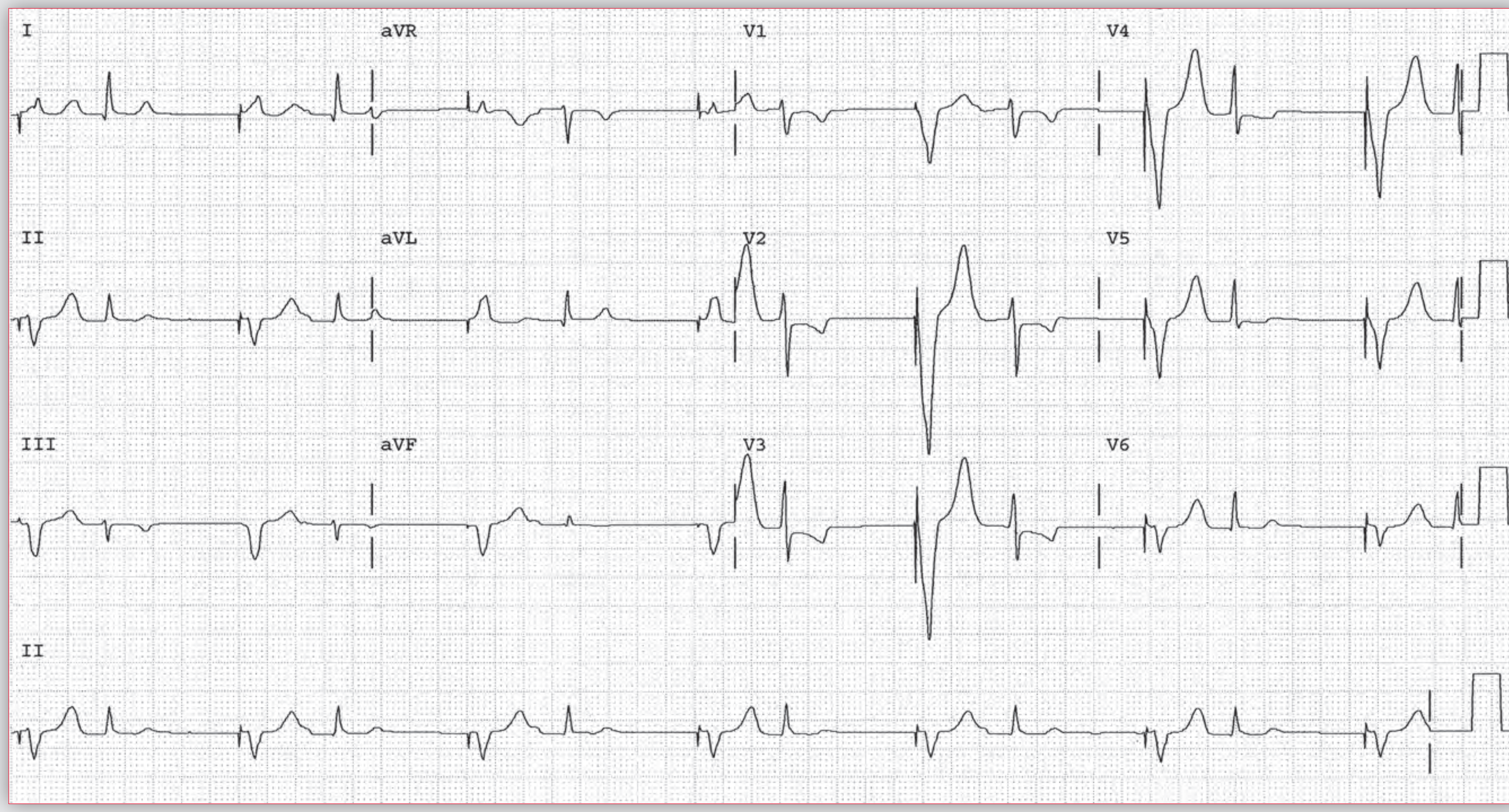
The rhythm is regular, although several intervals are slightly irregular (↔) at an average rate of 54 bpm. The first two and last QRS complexes (+) have a prolonged duration (0.16 sec) with a left bundle branch block morphology (except for a qR wave in V1 [*]), and there is a pacing spike or stimulus seen before each of these wide QRS complexes (+). The ventricular rate is 52 bpm. There are no obvious P waves seen, but in lead V1, there are irregular and rapid waveforms (↓). Hence the underlying rhythm is atrial fibrillation. QRS complexes 4, 5, and 7 (▲) do not have a pacemaker spike before them and the RR intervals are irregular (↔). They are slightly narrower than the first three QRS complexes (0.14 sec) and have a morphology of a typical right bundle branch block (RBBB) with an RSR′ morphology in V1 (←). These are native QRS complexes that are occurring at a rate faster than the lower rate limit of the pacemaker and hence sensed by the pacemaker and suppressing its output. The QT/QTc intervals of the native QRS complexes are normal (440/420 msec and 400/380 msec when corrected for the prolonged QRS complex duration). Complexes 3 and 6 (^) have the same width (0.14 sec) and morphology as the native QRS complexes, *ie*, a RBBB. Noted is a pacing stimulus (++) before these two complexes. However, the pacing stimulus appears to be slightly after the onset of the QRS complex. In addition, the QRS morphology and width of the third and sixth QRS complexes are different from the first two and last QRS complexes. Hence these are native QRS complexes, not resulting from the pacemaker, but which are occurring at the same rate as the pacemaker rate. There is a pacemaker stimulus occurring at the same time as native conduction occurs; this is called pseudofusion. Therefore, this is a demand ventricular pacemaker (*ie*, VVI). The seventh complex is a native QRS complex (↑) while the eighth complex is a result of the pacemaker impulse (+), hence it is a paced right ventricular complex.

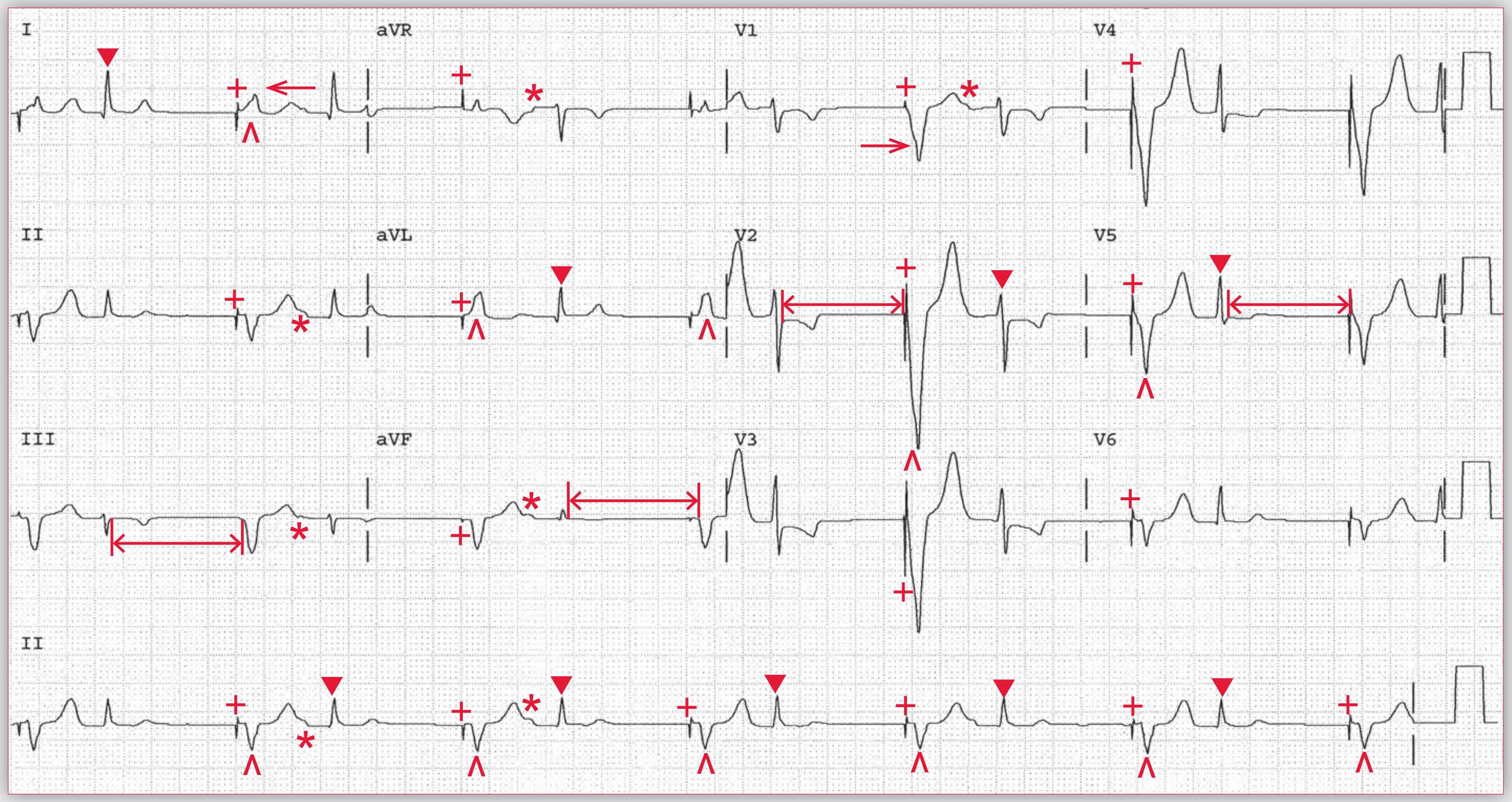
As the patient is in atrial fibrillation, the ventricular rate is irregularly irregular. Hence if the ventricular rate is faster than the lower limit of the pacemaker, the pacemaker output is inhibited. If the native ventricular rate falls below the lower rate limit of the pacemaker, there is a pacemaker output and a paced QRS complex. If there is consistent ventricular pacing (*ie*, VVI pacing), the rhythm will be regular. However, if the spontaneous ventricular rate is identical to the lower rate limited of the pacemaker, there will be a pacemaker stimulus occurring at the same time as the spontaneous QRS complex and the pacemaker stimulus will not result in ventricular capture. There is no pacemaker malfunction in this scenario. Pseudofusion can be avoided if the lower rate limit of the pacemaker is reduced or increased. ■

Notes

You note a regularly irregular rhythm when examining a patient and obtain the following ECG.

What is the rhythm diagnosis?





ECG 5 Analysis: Marked sinus bradycardia with first-degree AV block, demand right ventricular pacing (VVI)

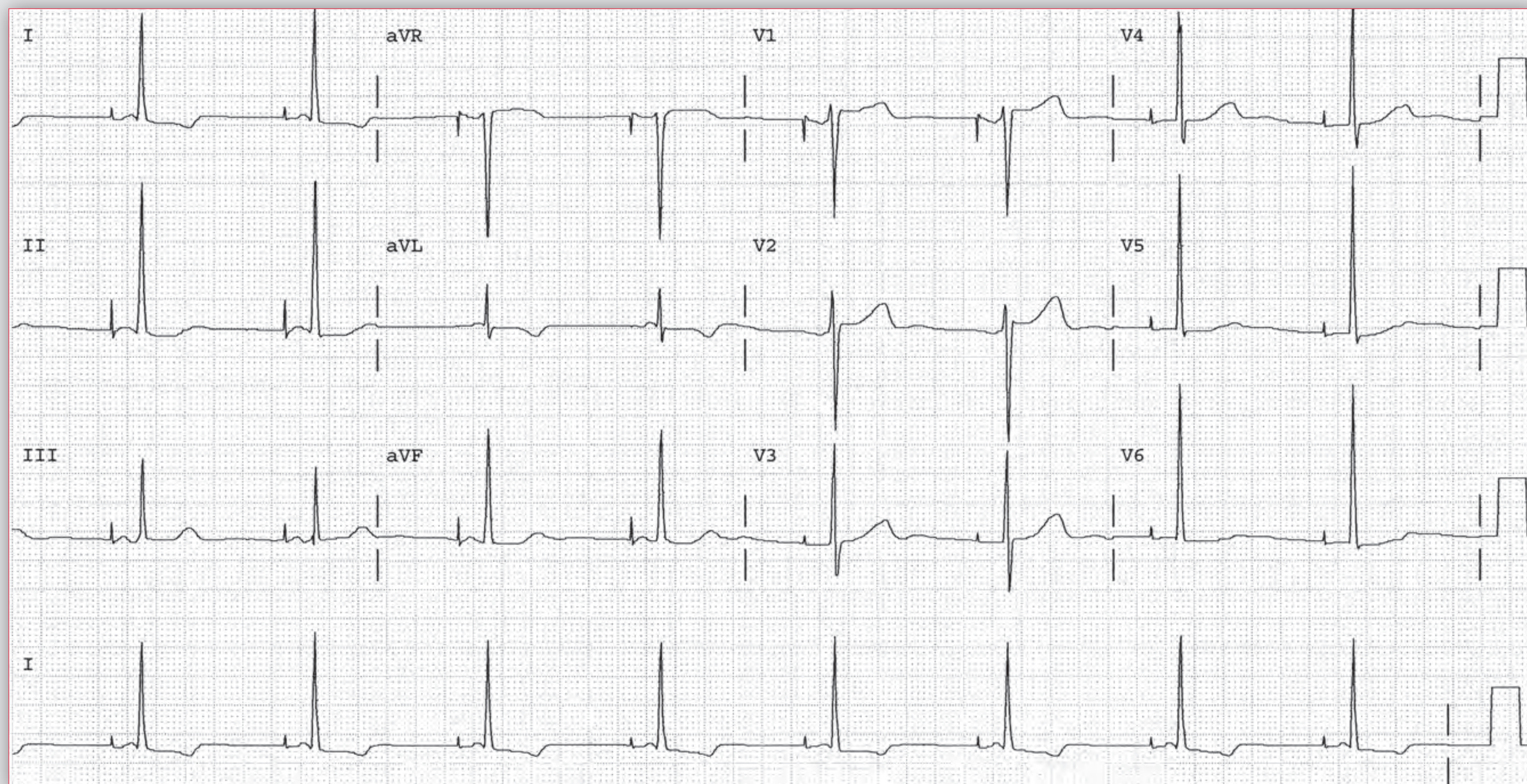
There are wide (0.18 sec) (^) and narrow (0.08 sec) (▼) QRS complexes. The wide complexes that have a left bundle branch block morphology (broad R wave in lead I [←] and QS complex in lead V1 [→]) are preceded by pacing stimuli (+). These are right ventricular paced complexes. The narrow complexes have a P wave before them (*) and the PR interval is stable (0.24 sec). The P wave is positive in leads I, II, and aVF. These are sinus complexes with a first-degree AV block or conduction delay. There is a fixed relationship between the sinus and the paced QRS complexes (↔) with a rate of 64 bpm. Hence this ECG shows sinus complexes followed by a sinus pause after which there is a ventricular paced complex that occurs at a rate of 64 bpm, the lower rate limit of the pacemaker. The pacemaker is a demand ventricular pacemaker or VVI mode. The QT/QTc intervals of the sinus complexes are normal (340/350 msec). ■

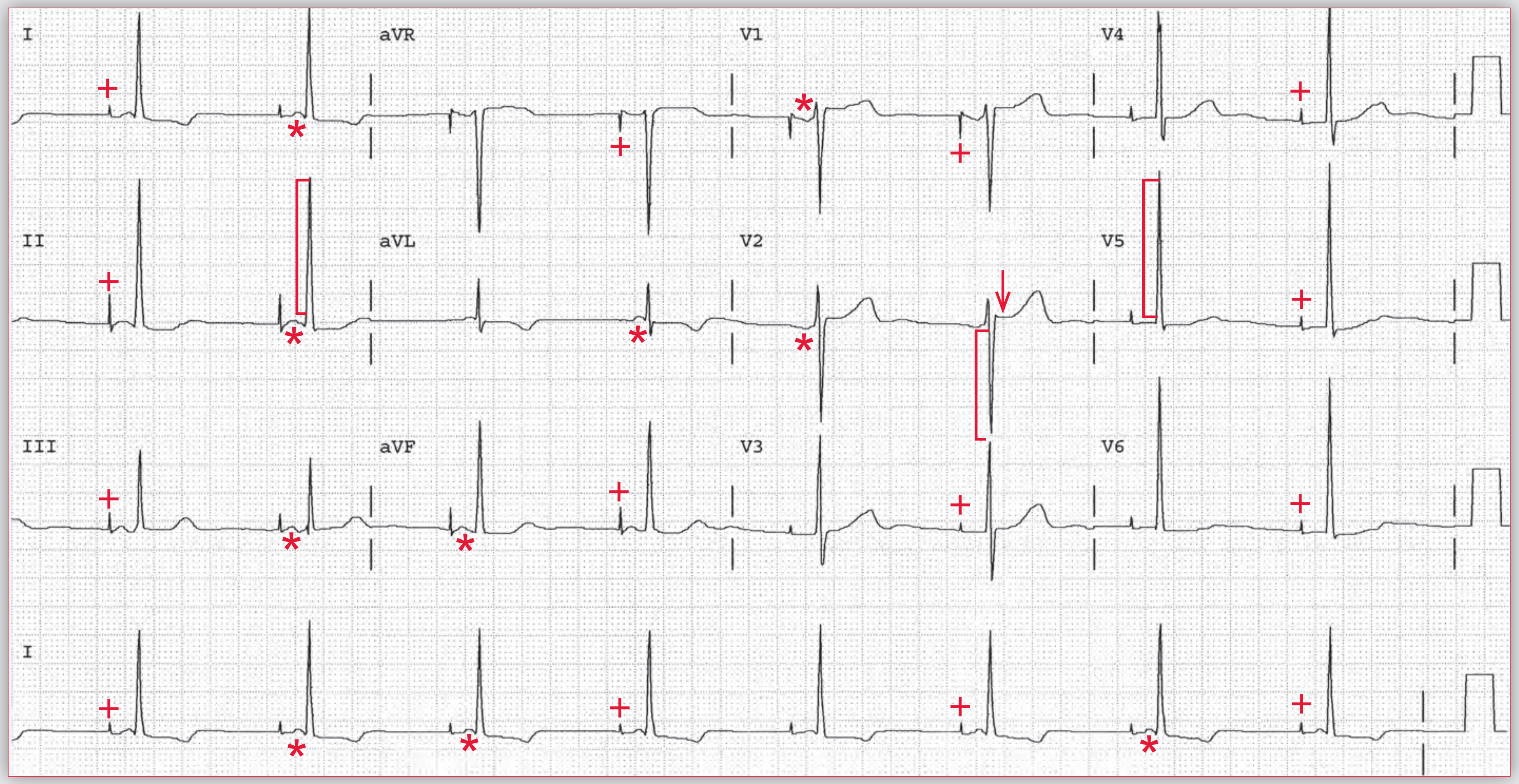
Notes

An 84-year-old woman with a history of heart failure with preserved ejection fraction, longstanding hypertension, and sick sinus syndrome presents to your clinic for routine follow-up. You obtain the following ECG.

Is the pacemaker functioning normally?

Is a revision of the pacemaker needed?





ECG 6 Analysis: Atrial pacing with native ventricular conduction, left ventricular hypertrophy, normal early repolarization

There is a regular rhythm at a rate of 50 bpm. A pacing stimulus, artifact, or spike (+) is seen before each P wave (*), indicating that this is atrial pacing. Since the P wave is initiated by a pacemaker, the P-wave axis and morphology may be abnormal, related to the location of the atrial lead. Abnormalities of the right or left atrium cannot be established, as the P wave is abnormal due to its initiation by a pacemaker stimulus and hence an abnormal atrial activation sequence.

There is a QRS complex that follows each paced P wave and the PR interval is stable (0.16 sec). The QRS complex duration is normal (0.08 sec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/400 msec). The QRS morphology is normal, but the R waves have an increased amplitude ([ ), *ie*, 25 mm in lead II, 28 mm in V5 and 20 mm S wave in lead V2, meeting criteria for left ventricular hypertrophy (*ie*, > 20 mm in any limb lead and SV2 + RV5 ≥ 35 mm. There is also slight J point and ST-segment elevation in lead V2 (↓) that is early repolarization.

There is 100% atrial capture and hence the pacemaker function appears to be normal. The PR interval is normal and consistent, suggesting intact AV nodal conduction. As the patient has a history of a sick sinus syndrome, it appears that she has only sinus node dysfunction without any involvement of the AV node. Indeed, she likely has significant underlying sinus bradycardia since she is atrial pacing at a rate of only 50 bpm and here spontaneous sinus rate must be less than this. Hence an atrial pacemaker is an appropriate therapy in this situation and there is no need for ventricular pacing. ■

Notes

A 62-year-old woman comes for routine check of her single-chamber pacemaker. She is complaining of intermittent lightheadedness. You note that on her prior ECG, she had a complete left bundle branch block.

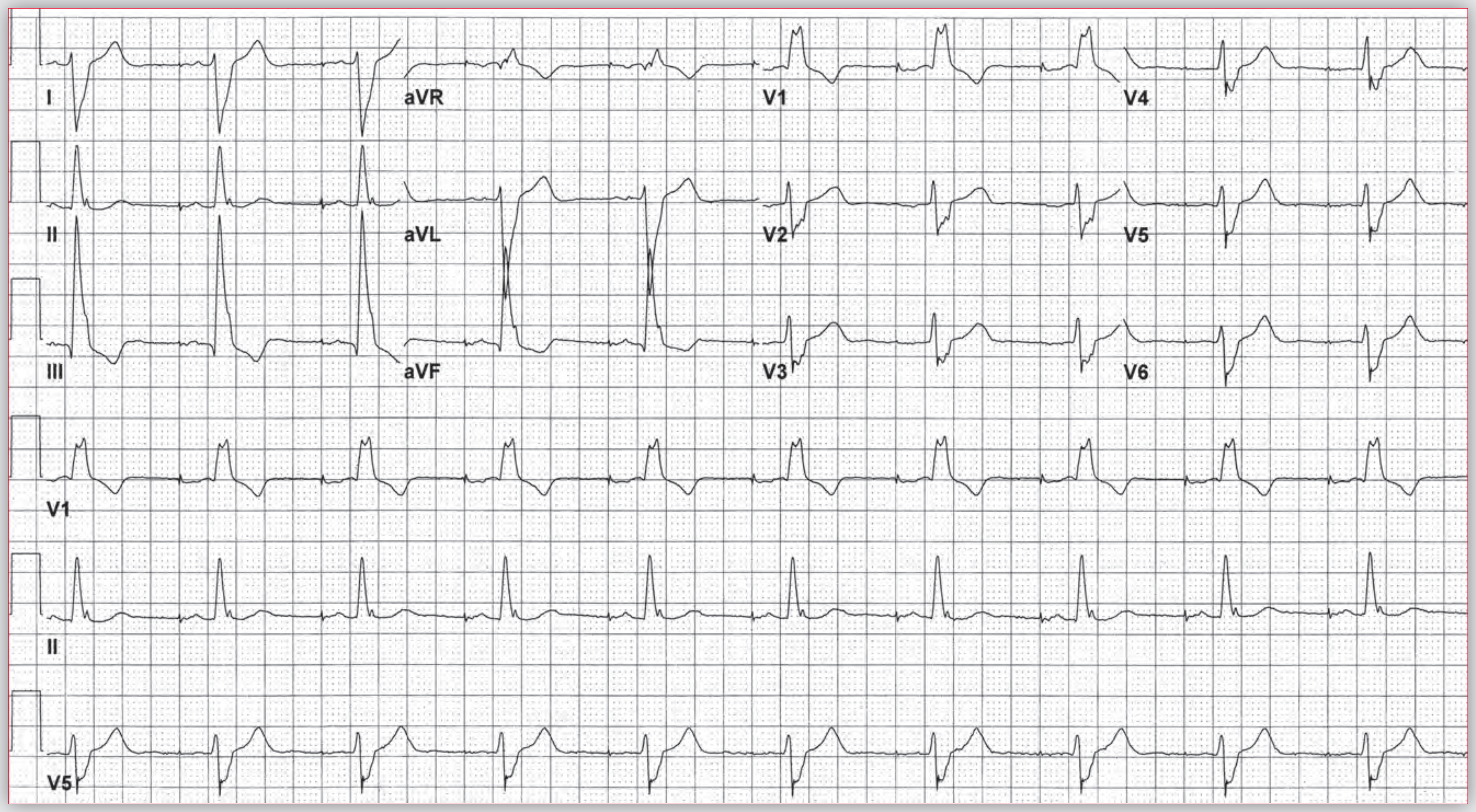
What is your next step in management?

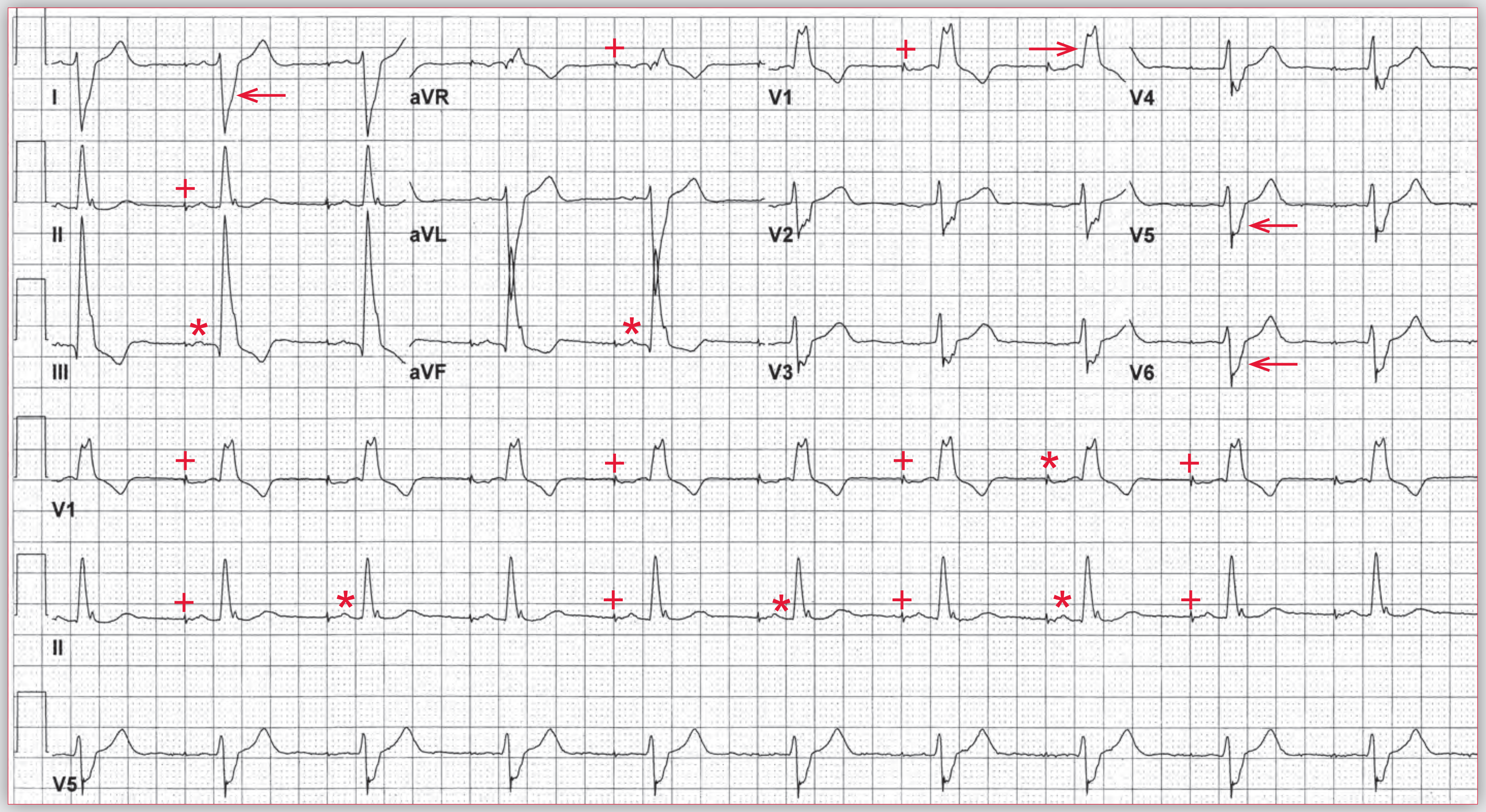
(A) Electrophysiology study (EPS)

(B) Increase sensitivity of pacemaker lead

(C) Add a right ventricular lead

(D) No further treatment is required





ECG 7 Analysis: Atrial pacing with native ventricular conduction, first-degree AV block, RBBB, left posterior fascicular block (bifascicular block)

There is a regular rhythm at a rate of 60 bpm. A pacing stimulus, artifact, or spike (+) is seen before each P wave (*), indicating that this is atrial pacing. Since the P wave is initiated by a pacemaker stimulus, P wave axis and morphology may be abnormal, related to the location of the atrial lead. Abnormalities of the right or left atrium cannot be diagnosed, as the sequence of atrial activation is abnormal as a result of the paced P wave.

A nonpaced native QRS complex follows each paced P wave, and the PR interval is stable but prolonged (0.24 sec) as a result of a first-degree AV block. The QRS complex duration is prolonged (0.16 sec), and it has a morphology of a typical right bundle branch block (RBBB) (broad R wave in V1 [→] and broad S waves in leads I and V5–V6) [←]). The axis is rightward between +90° and +180° (negative QRS complex in lead I and positive in lead aVF). The rightward axis is not the result of any definable etiology, such a lateral wall infarction, Wolff-Parkinson-White pattern, R-L arm lead switch, dextrocardia, or right ventricular hypertrophy. It is due to a left posterior fascicular block (LPFB). The QT/QTc intervals are normal (440/440 msec and 380/380 msec when the prolonged QRS complex duration is considered). The presence of a RBBB and a LPFB is considered to be bifascicular block. The fact that there is also a first-degree AV block present does not necessarily indicate trifascicular disease, as the prolonged PR interval may be the result of slow conduction through the AV node or the His-Purkinje system (*ie*, the left anterior fascicle is the remaining fascicle that is conducting).

We are told that the patient has a single-chamber pacemaker, so she has a single lead in the right atrium given the atrial pacing that is observed. Her native ventricular conduction consists of bifascicular block with RBBB and LPFB. A prior ECG is reported to exhibit left bundle branch block (LBBB). Thus, the patient exhibits intermittent RBBB and LBBB, which is diagnostic of bibundle branch block (or trifascicular disease). The finding of disease of both bundles when associated with symptoms meets class I criteria for ventricular pacemaker (answer C). In this situation, the symptoms are suggestive of intermittent complete heart block (episodic lightheadedness).

In the setting of chronic bifascicular or trifascicular block, the class I indications for permanent pacing include: (1) intermittent complete heart block; (2) type II second-degree AV block; and (3) alternating bundle branch block. There is no need for an EPS in this setting since a class I criteria for ventricular pacing has already been established. However, in the absence of such criteria, an EPS can be performed, and if the HV interval (*ie*, a measure of His-Purkinje conduction time) is > 100 msec, this represents a class IIa indication for ventricular pacing in asymptomatic individuals. There is nothing on the ECG that suggests reduced pacemaker sensitivity, as pacemaker function appears to be normal, although its sensing function cannot be established as the rhythm is 100% atrially paced and there are no spontaneous atrial complexes. ■

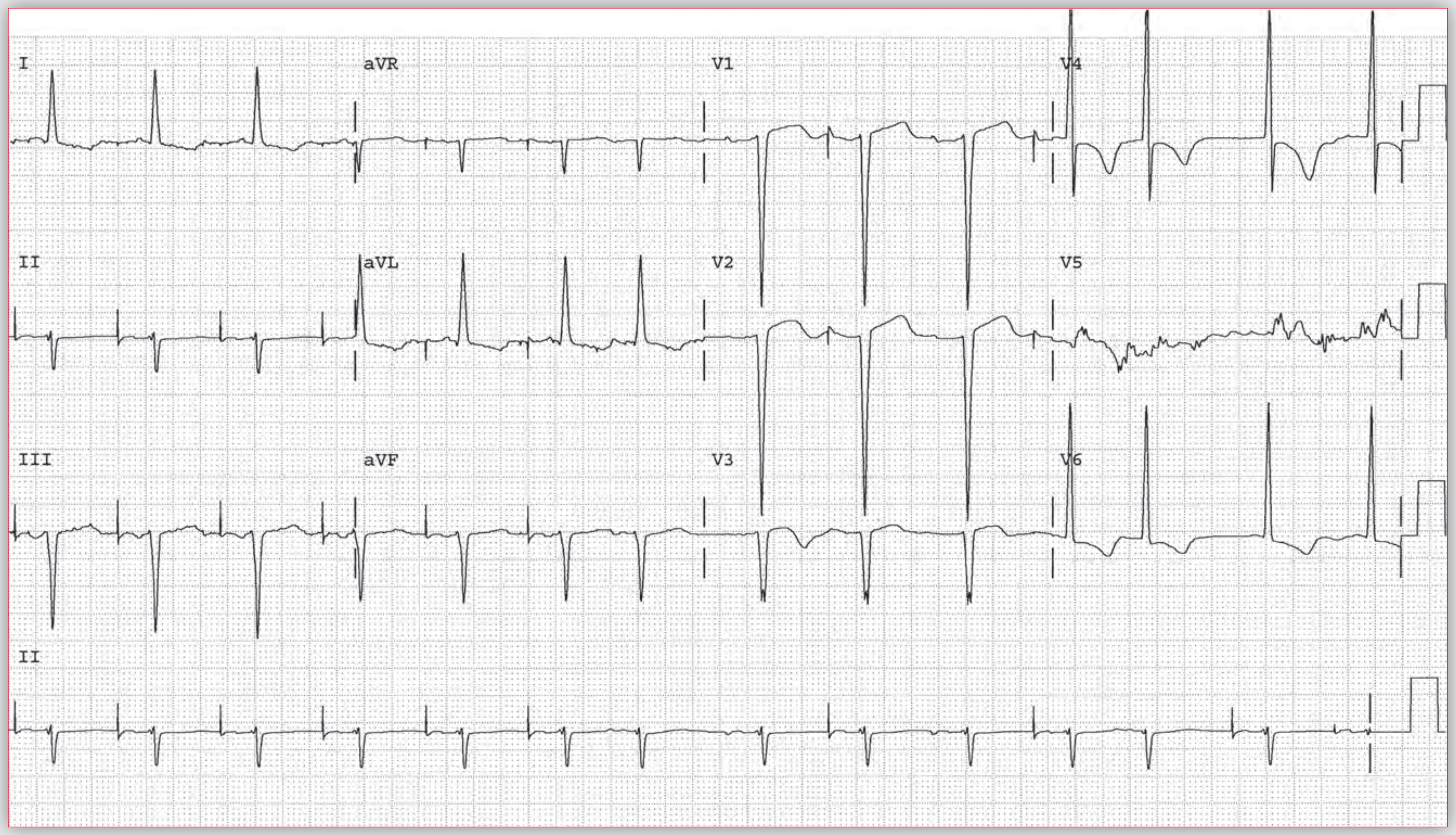
Notes

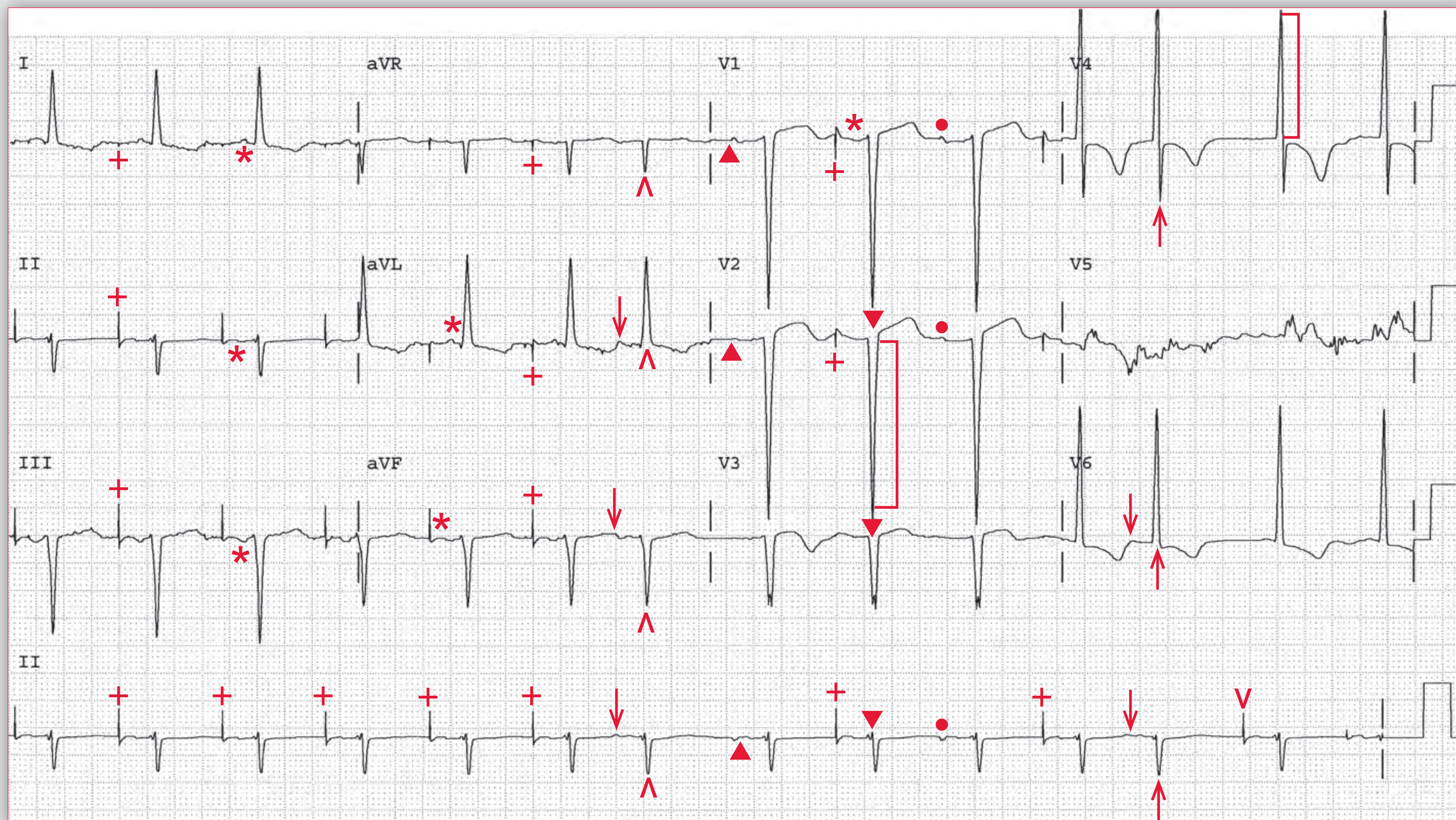
A 70-year-old woman with a history of sinus node dysfunction for which she received a pacemaker comes to the emergency department because of an irregular pulse. She is concerned that her pacemaker is not functioning properly.

What is her rhythm?

Is there a problem with her pacemaker?

Is any therapy necessary?





ECG 8 Analysis: Atrial paced rhythm, first-degree AV block, premature atrial complexes, sinus complex, left anterior fascicular block, chronic anteroseptal myocardial infarction, left ventricular hypertrophy with nonspecific ST-T wave changes

There is a basically a regular rhythm at a rate of 82 bpm. There is a pacing stimulus, artifact or impulse (+) prior to each P wave (*); hence this is an atrially paced rhythm and the lower limit of the pacemaker is 82 bpm. Since the P wave is initiated by a pacemaker, P-wave axis and morphology are often abnormal, related to the location of the atrial lead. The presence of atrial hypertrophy or an atrial abnormality cannot be established as atrial activation is abnormal. The seventh and twelfth QRS complexes (^, ↑) are early. They are preceded by a P wave (↓) but no pacemaker stimulus, and the QRS morphology is the same as the native QRS complexes. The PR interval before both of these premature complexes is the same (0.22 sec). These are premature atrial complexes. After the seventh complex, there is a pause, and then there is a spontaneous P wave (▲) occurring at a rate of 90 bpm, which is at a faster rate than the lower rate limit of the pacemaker (*ie*, 82 bpm). Hence there is no atrial pacemaker stimulus seen as the atrial output is suppressed. Following this QRS complex there is an appropriately timed atrial stimulus and an atrial paced QRS complex (complex 9 [▼]). Following the ninth QRS complex there is another spontaneous P wave (●) without a preceding pacemaker stimulus that occurs at a rate of 90 bpm and also suppresses the atrial pacemaker output. The eleventh QRS complex is again atrially paced. The twelfth QRS complex (↑) is also preceded by a spontaneous and early P wave (↓). This is also a premature atrial complex. After the twelfth QRS complex (↑) the pause is ended by an atrial pacemaker impulse (v) as there was no spontaneous sinus or atrial impulse before the pacemaker output occurred at its pacing rate of 82 bpm.

There is a QRS complex after each paced P wave and the PR interval is stable (0.26 sec). Hence there is a first-degree AV block (or prolonged AV conduction). The QRS complex duration is normal (0.08 sec) and there is a normal morphology, although the QRS amplitude is increased ( ]) (R wave in lead V4 = 26 mm and S wave in V2 = 33 mm) diagnostic of left ventricular hypertrophy (*ie*, SV2 + R4 ≥ 35 mm). There are ST-T wave changes seen in leads I, aVL, V4 and V6, which are secondary to left ventricular hypertrophy. The axis is extremely leftward between –30° and –90° (QRS positive in lead I and QRS negative in leads II and aVF); as the QRS complex morphology is rS, this is a left anterior fascicular block. Lastly there are no R waves in leads V1–V3 (*ie*, QS complexes), consistent with an anterior septal myocardial infarction. The QT/QTc intervals are normal (360 msec/420 msec).

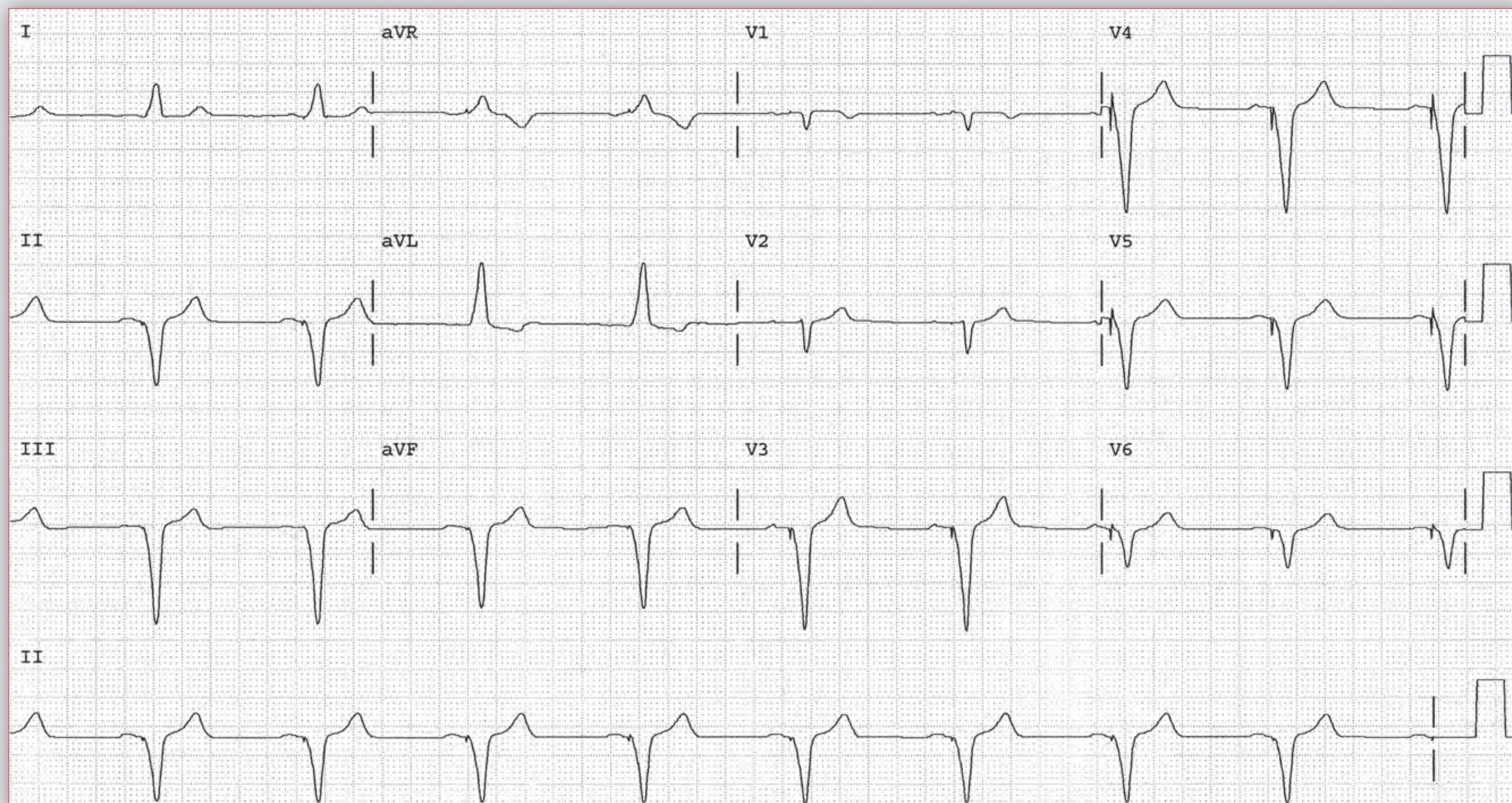
Hence this is a demand atrial pacemaker, functioning in an AAI mode. Its function is normal and hence no additional therapy is necessary. ■

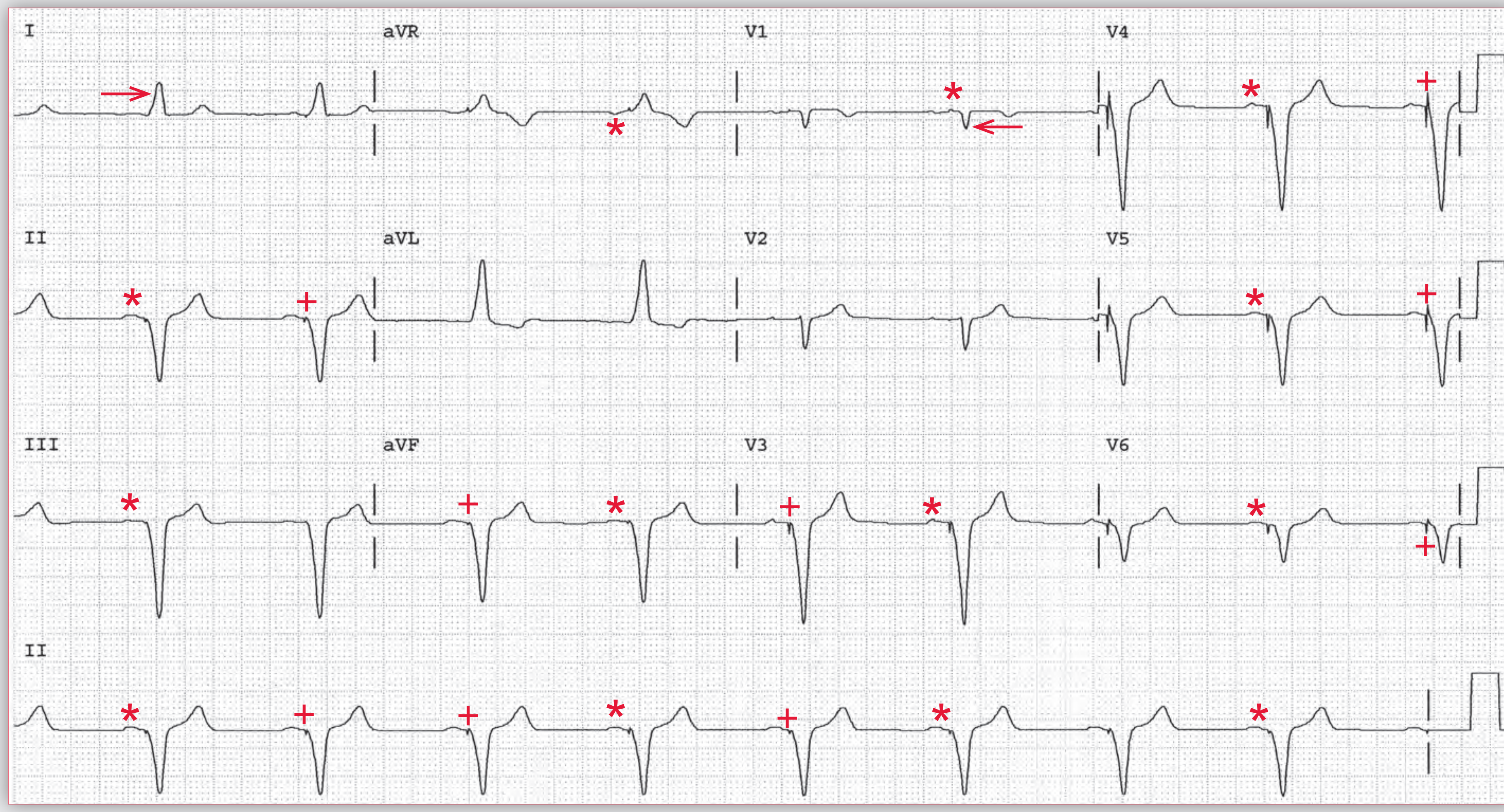
Notes

A 72-year-old man is seen for a first visit with a new primary care provider. He states that he had a pacemaker inserted three years ago, but does not know any other details. He denies any previous symptoms and does not know why he had a pacemaker. An ECG is obtained.

What type of pacemaker was inserted?

How many leads are present?





ECG 9 Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed right ventricular paced rhythm (P-wave synchronous ventricular pacing)

There is a regular rhythm at a rate of 50 bpm. There is a P wave (*) seen before each QRS complex. The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus P wave. Following each P wave is a pacemaker artifact, stimulus, spike, or impulse (+) with stable PR interval (or AV delay) of 0.20 sec. After each pacemaker stimulus, there is a QRS complex that has a prolonged duration (0.16 sec) and has a left bundle branch morphology with a broad R wave in lead I (→) and a QS complex in lead V1 (←). Hence the pacemaker lead is in the right ventricle. The QT/QTc intervals are normal (440/400 msec and 380/350 msec when the prolonged QRS complex duration is considered).

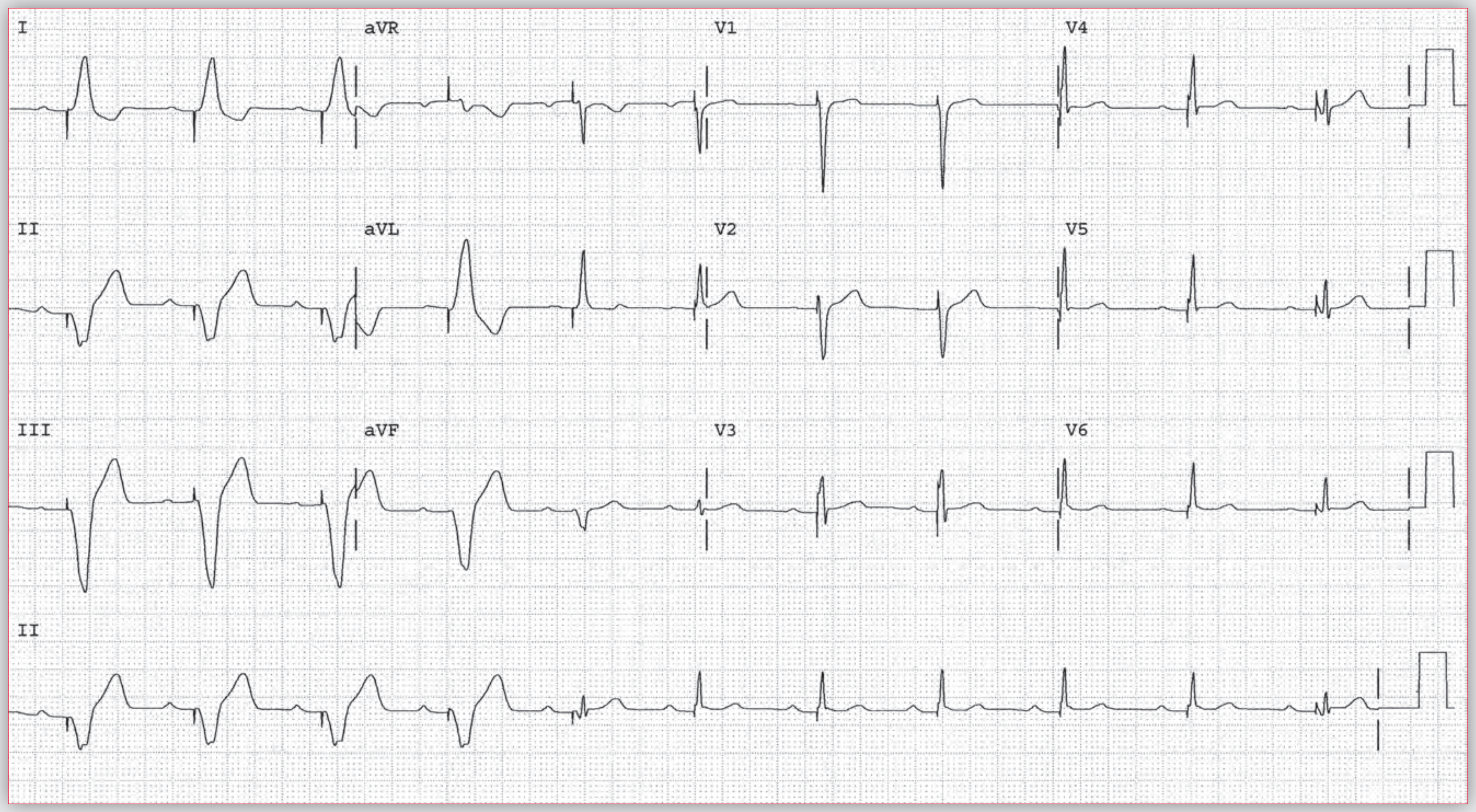
Although there is an extreme leftward axis between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF), this is not a left anterior fascicular block as the QRS complex results from ventricular pacing, which directly activates the ventricular myocardium, bypassing the normal His-Purkinje system. The pacemaker is functioning in an atrial (P wave) sensed ventricular paced mode (also known as P-wave synchronous right ventricular pacing). Therefore, this is a dual-chamber pacemaker and there is a second pacemaker electrode in the right atrium that is sensing spontaneous atrial activity. Therefore, it is likely that the reason for the pacemaker was AV block, although it is not certain what degree of AV block was present. ■

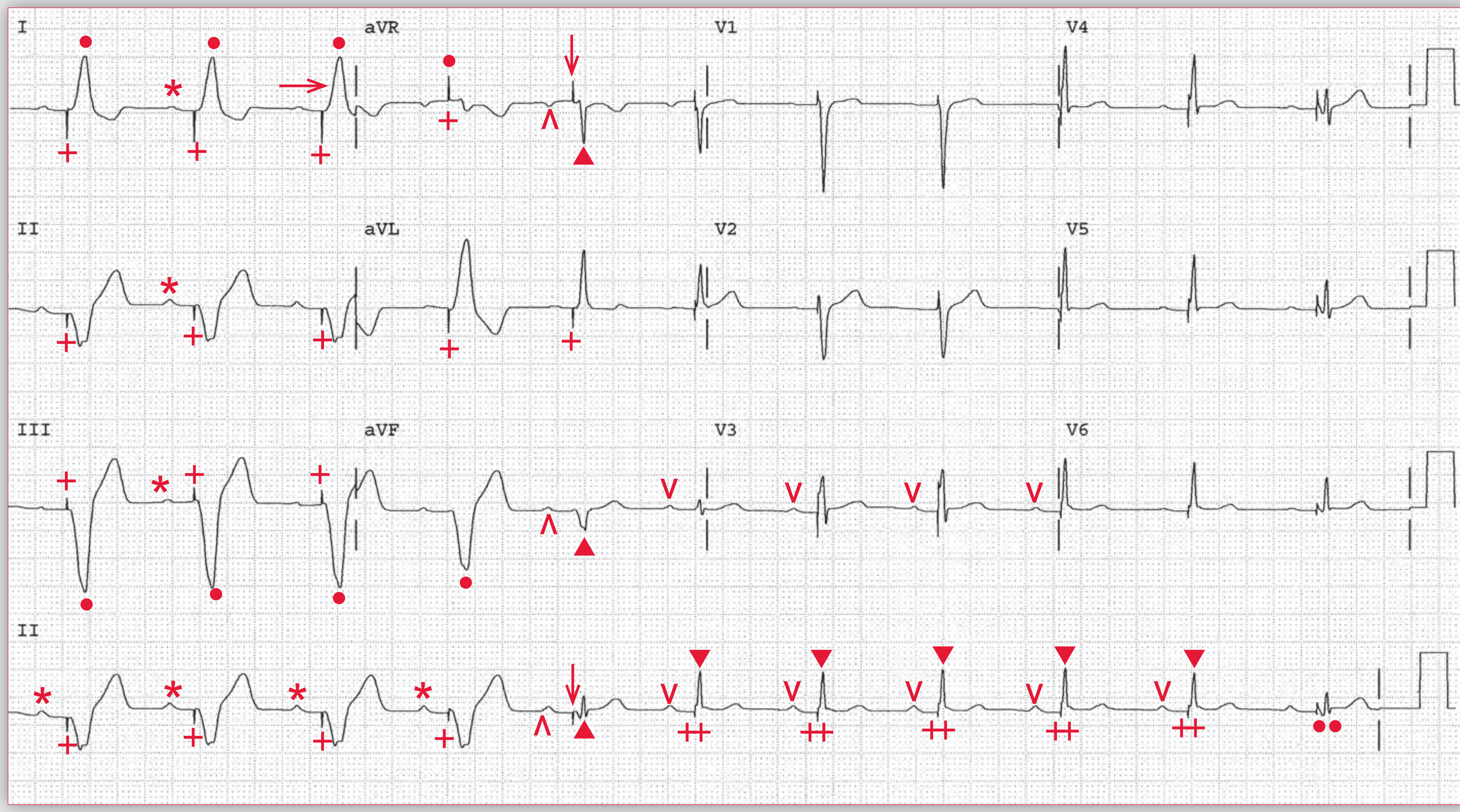
Notes

Core Case 10

An 80-year-old woman with a history of a dilated cardiomyopathy and a pacemaker is seen in the office with complaints of shortness of breath, which has worsened over the past week. On physical examination, she has bilateral rales, a third heart sound, a murmur of mitral regurgitation, and bilateral lower-extremity edema. An ECG is obtained and admission to the hospital is advised.

What type of pacemaker does this patient have, and is it functioning normally?





ECG 10 Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous right ventricular pacing), pseudofusion

The first four QRS complexes (●) are regular at a rate of 64 bpm. The QRS complex is wide (0.18 sec) with a left bundle branch block (LBBB) morphology (broad R wave in lead I [→]), and there is a pacing spike or stimulus (+) before each of these QRS complexes; hence there is right ventricular pacing. There is a P wave (*) before each QRS complex with a stable PR interval (or AV delay) at 0.26 sec. The P wave is positive in leads I, II, aVF, and V4–V6. Hence there is a sinus rhythm with a first-degree AV block or prolonged AV conduction. The patient has a dual-chamber pacemaker with a pacing lead in the right atrium and right ventricle. It is functioning as a P-wave synchronous or atrial activated ventricular pacing. There is a P wave (^) and pacing spike or stimulus (↓) in front of the fifth QRS complex (▲). The PR interval (AV delay) is 0.26 sec, identical to the first three complexes. The QRS complex duration (0.10 sec) and morphology are different from the first four paced QRS complexes. Hence this is a fusion complex, meaning that the AV delay of the pacemaker is slightly shorter than the intrinsic PR interval. Hence there is initial left ventricular activation from the pacemaker fusing with left ventricular activation via the AV node–His-Purkinje system.

The sixth through the tenth QRS complexes (▼) are regular at a rate of 68 bpm. They have a narrow and normal QRS duration (0.08 sec). There is a P wave (v) before each QRS complex with a stable PR interval (0.26 sec). The QT/QTc intervals are normal (340 msec/360 msec). Noted in front of each QRS complex is a pacemaker spike or stimulus (++); however the pacemaker impulse does not result in a paced QRS complex as it does not capture the ventricle; rather, it is a native or normal QRS complex that does not have a LBBB morphology. This is termed pseudofusion and is due to the fact that the intrinsic PR interval is identical to the AV delay of the pacemaker. Hence there is left ventricular activation via the normal conduction system that occurs at the same time as activation via the pacemaker; the pacemaker stimulus is not suppressed, but it does not capture the ventricular myocardium. The last QRS complex (●●), which has a pacemaker stimulus before it, is similar in morphology to the fourth QRS complex and is a fusion complex.

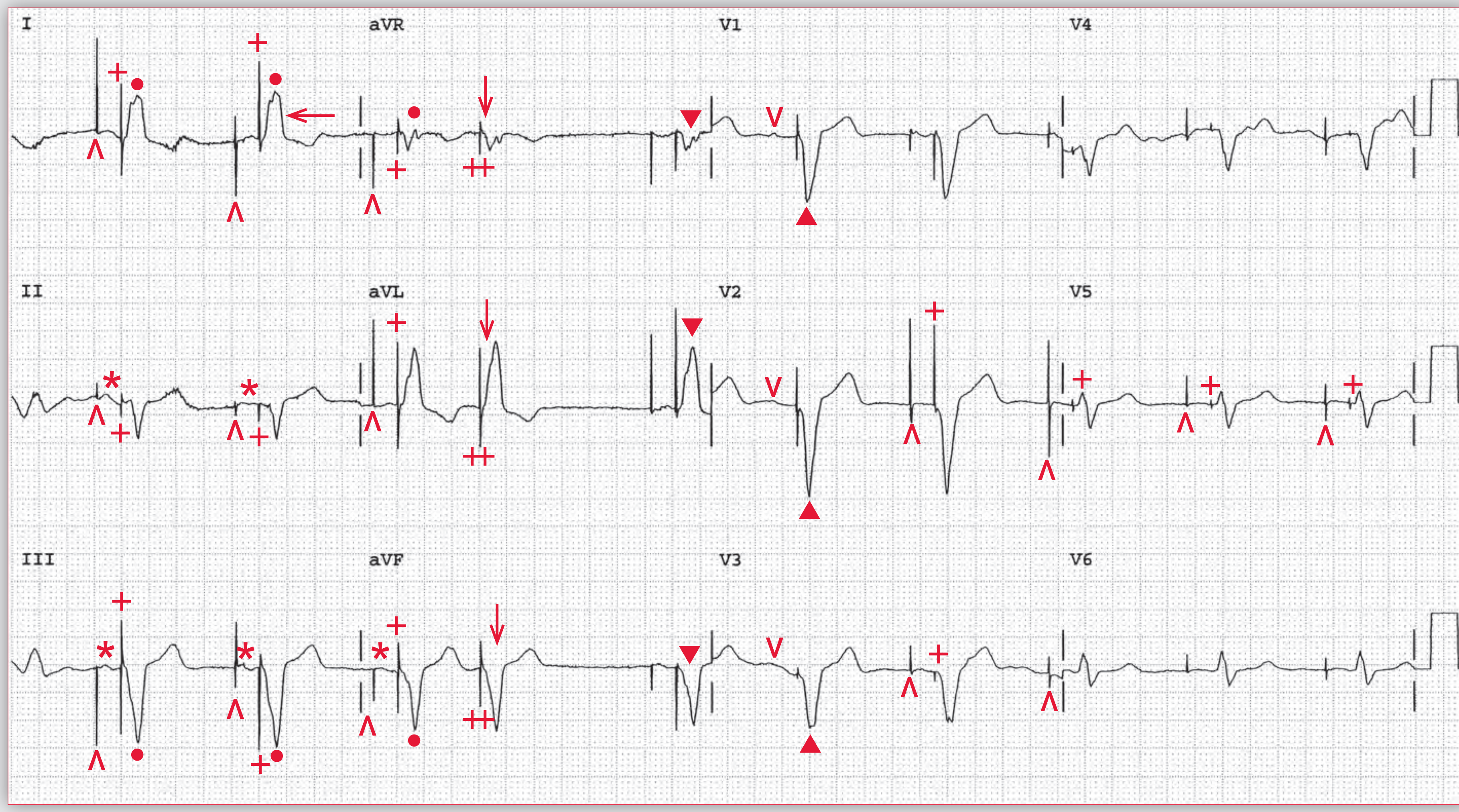
The pacemaker is functioning normally. Based on the ECG findings, this patient has transient AV block. With the development of the AV block, there is appropriate ventricular pacing, based on the AV delay of the pacemaker (*ie*, 0.26 sec). When AV conduction is restored, with an intrinsic PR interval of 0.26 sec, there is pseudofusion or more pronounced fusion, as seen with complex 4 and the last QRS complex, which means than the intrinsic PR interval is slightly longer than 0.26 sec. This does not represent a problem with the pacemaker or the lead. When there is complete ventricular capture, *ie*, the first three complexes, it is unclear what degree of AV block is present (*ie*, it may be a markedly prolonged PR interval > 0.26 sec, second- or third-degree AV block). ■

Notes

An 80-year-old man with a history of a sick sinus syndrome for which a pacemaker was inserted four years ago presents to the emergency department with complaints of palpitations that have been occurring more frequently. He denies other cardiac symptoms, although he occasionally feels dizzy. Physical examination is normal. An ECG is obtained.

Does the ECG suggest a cause for the symptoms?

What other studies would be helpful?



ECG 11 Analysis: Dual-chamber pacemaker, AV sequential pacing, premature atrial complex

There is a paced rhythm. The first 3 QRS complexes (●) that have a left bundle branch block morphology (QRS complex duration 0.18 sec with broad R wave in lead I [←]), have a pacemaker spike, artifact, or stimulus (^) before the P wave (*) and also in front of the QRS complex (+). The AV delay is 0.18 sec. Hence this is a dual-chamber pacemaker, functioning in an AV sequential mode at a rate of 60 bpm. The fourth QRS complex (↓) is early and there is a pacing stimulus (++) only in front of the QRS complex. Although there is no obvious atrial activity seen before this early QRS complex, the ventricular stimulus is premature and hence must be in response to atrial activity. Therefore this is a premature atrial complex that results in a ventricular paced complex (atrial sensed, ventricular paced). After the pause (which is identical to the lower rate limit of the pacemaker, *ie*, 60 bpm), there is an AV sequentially paced complex (▼). The sixth QRS complex (▲) has a native P wave (v), without an atrial pacemaker stimulus, followed by a ventricular paced complex. This is due to the fact that the native P wave occurs at a faster rate (64 bpm) than the lower rate of the pacemaker. This represents P-wave activated or synchronous ventricular pacing. The last four complexes are also AV sequentially paced (^,+). The QT/QTc intervals are prolonged (460/460 msec) but are normal when the prolonged QRS complex duration is considered (380/380 msec).

The ECG shows that the pacemaker function is normal.

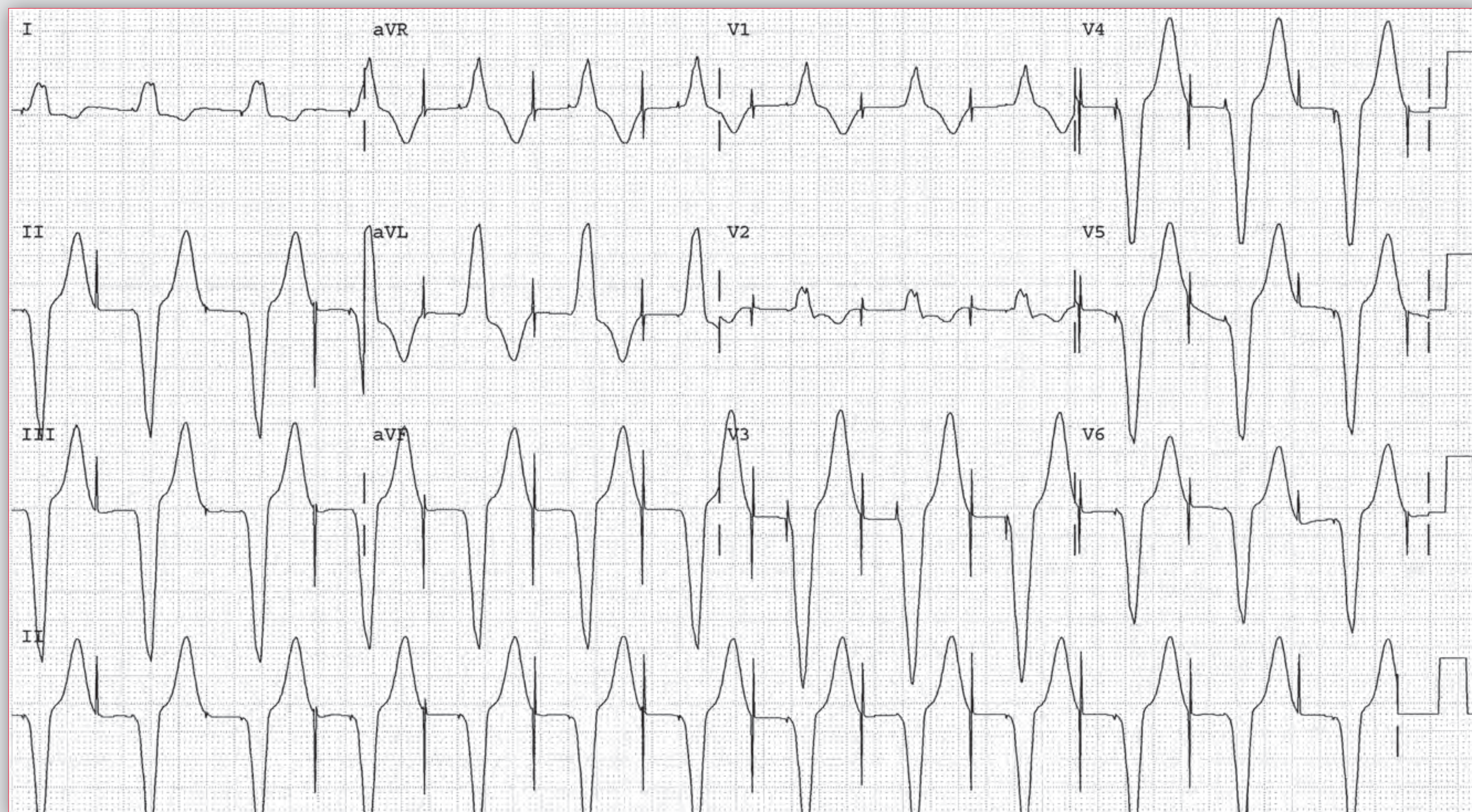
The ECG does not show any specific abnormality that would be responsible for the symptoms. Premature atrial complexes are common and are not likely to be the cause of symptoms. However, they might be responsible for provoking other atrial arrhythmias that if sustained might be associated with symptoms of palpitations. Hence prolonged monitoring, such as with an event recorder, would be useful to establish the presence of a prolonged atrial tachyarrhythmia. ■

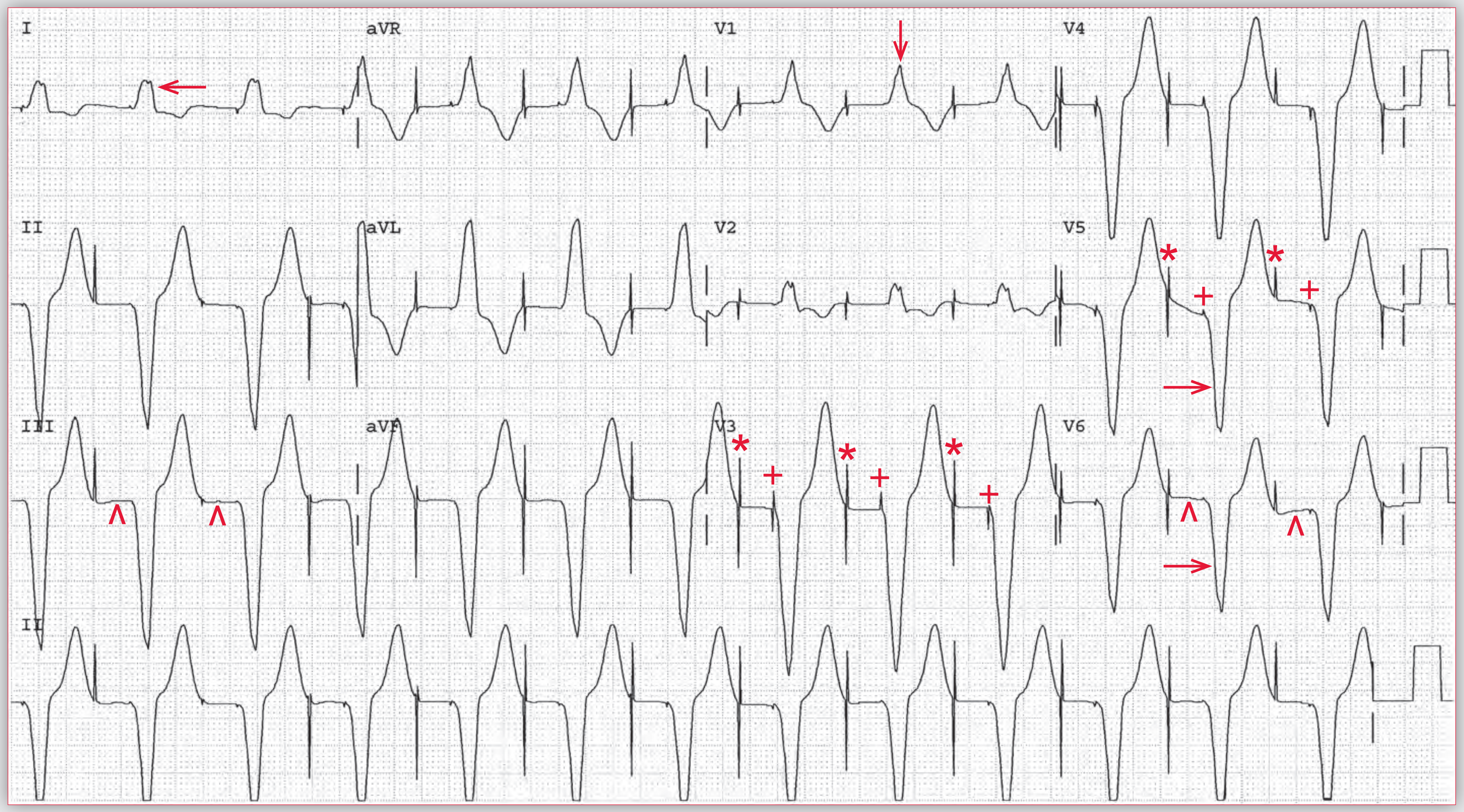
Notes

A 68-year-old man with a history of an ischemic cardiomyopathy presents with symptoms of chest discomfort, occurring with exercise. His physical examination is unremarkable. An ECG is obtained.

What is the mode of pacing?

Does the pacemaker require any adjustment?





ECG 12 Analysis: Dual-chamber pacemaker, AV sequential pacing

There is a regular rhythm at a rate of 80 bpm. There are two pacing stimuli seen: the first (*) is an atrial stimulus and the second is a ventricular stimulus that is in front of each QRS complex (+). The QRS complex duration is 0.18 sec, and the QT/QTc intervals are normal (380/440 msec and 300/350 msec). Hence this is a dual-chamber pacemaker functioning in an AV sequential mode. The AV delay of the pacemaker is 0.26 sec. However, there are no obvious P waves or atrial activity seen and thus it is not certain if there is atrial capture. However, there appears to be a small P wave after the atrial stimulus in leads III and V6 (^). The QRS complex has a left bundle branch block morphology with a broad R wave in lead I (←) and a QS complex in leads V5–V6 (→). However, there is a tall broad R wave in lead V1 (↓), which may be seen if the lead is against the septum; hence the ventricular lead is in the right ventricle.

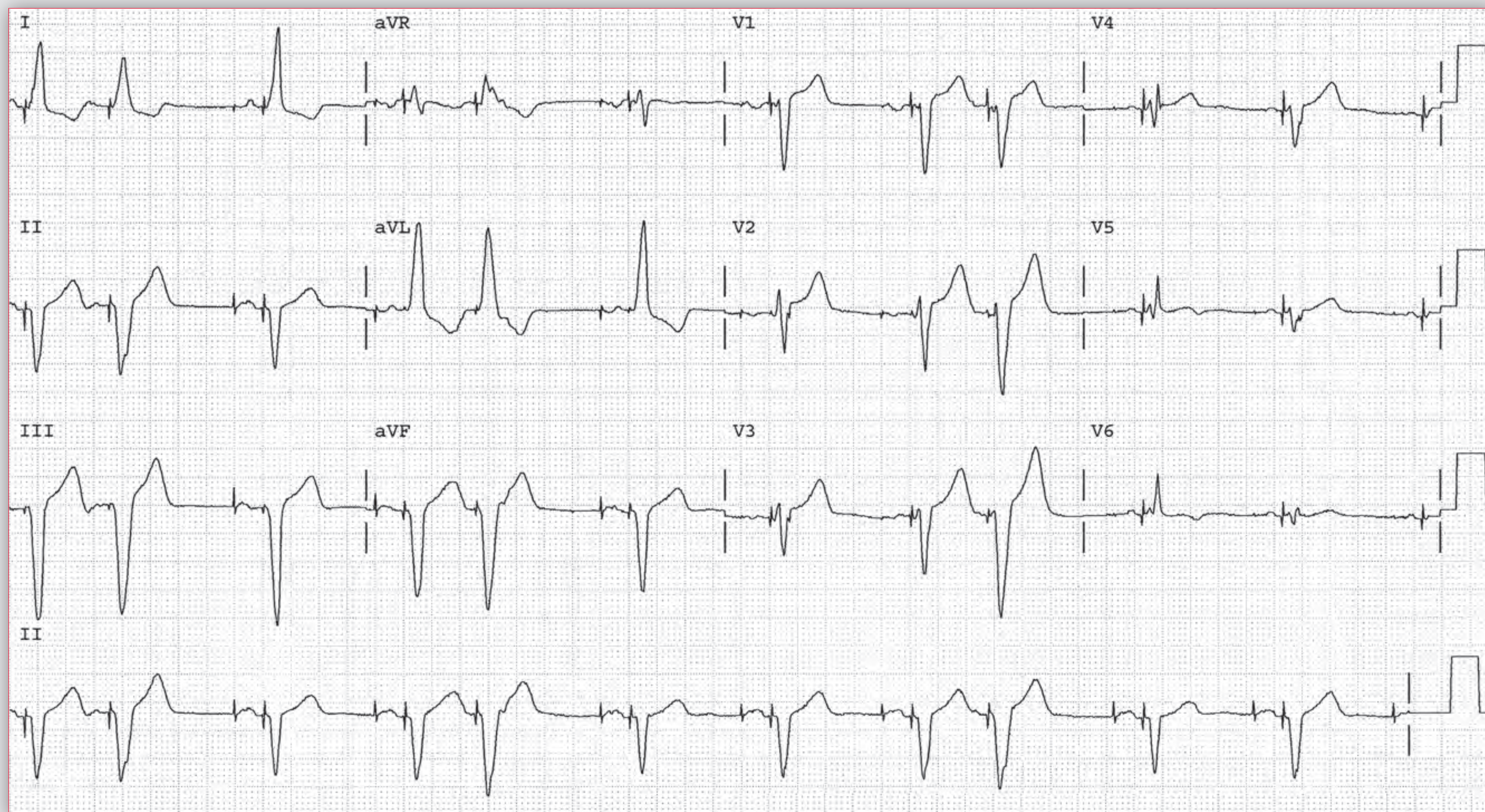
Of concern is the fact that the rate of the pacemaker is 80 bpm. As there is AV sequential pacing, the pacemaker is not tracking atrial activity. Therefore, the lower rate limit of the pacemaker is 80 bpm. For this patient, who is complaining of symptoms suggesting angina, therapy with a β-blocker, which is often a first-line therapy for angina, will not result in slowing of the heart rate, and hence this medication is not likely to be effective for symptom relief. Therefore, it would be useful to lower the rate limit of the pacemaker, which by resulting in a slower heart rate would be useful for therapy of angina. Reducing the lower rate limit of the pacemaker might result in spontaneous sinus rhythm. If so, a β-blocker therapy could be initiated so as to keep the heart rate slow, although this would be limited by the atrial pacing rate (*ie*, the lower rate limit of the pacemaker). ■

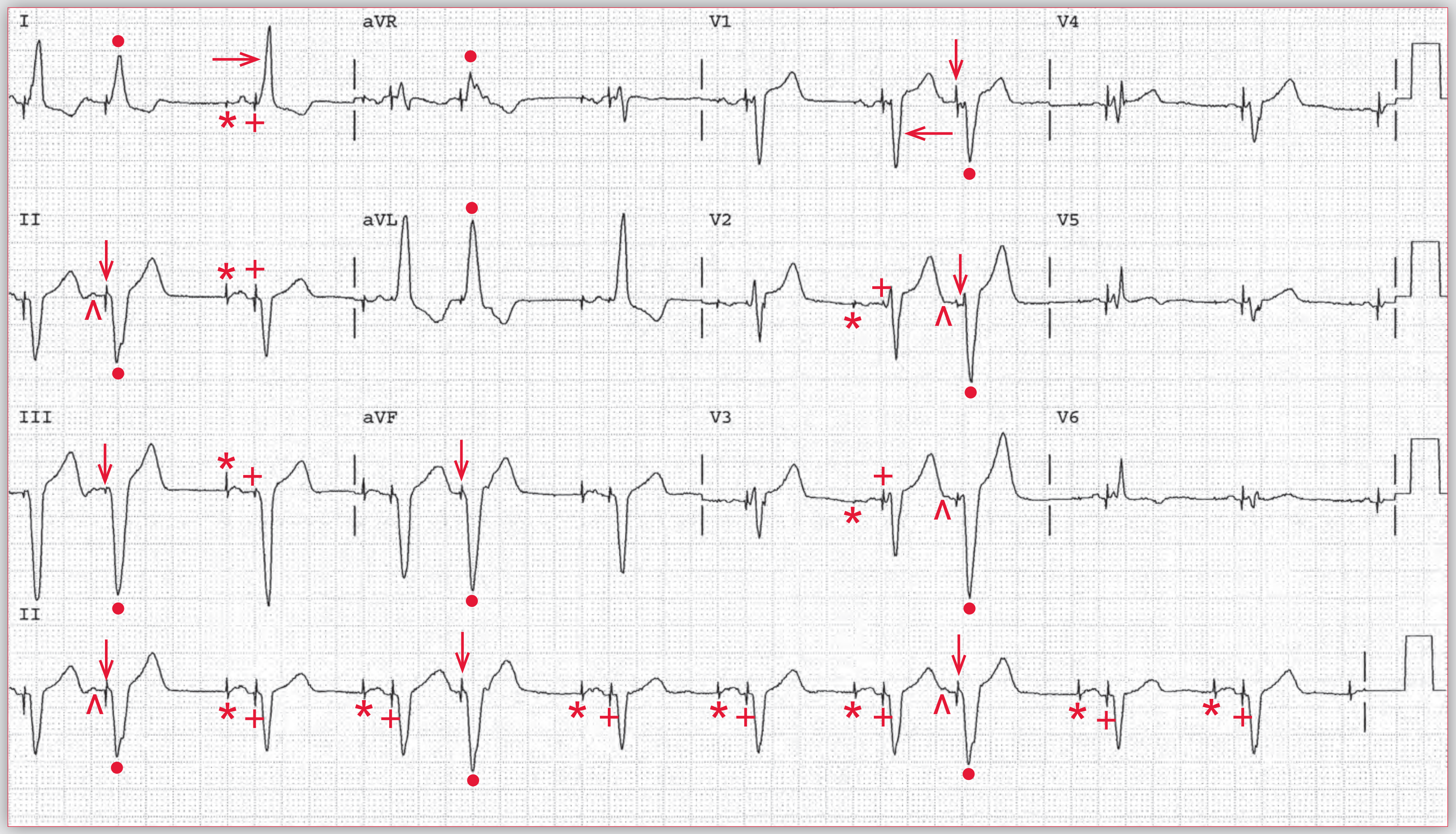
Notes

A 74-year-old man with a history of syncope determined to be due to complete heart block requiring pacemaker insertion is seen by his cardiologist and is complaining of an irregular pulse rate. He is alarmed by this because his pulse rate in the past was always regular, and he is concerned that there is a problem with the pacemaker.

Is the pacemaker functioning normally?

Is any further therapy necessary?





ECG 13 Analysis: Dual-chamber pacemaker, AV sequential pacing, premature atrial complexes

There are pacemaker stimuli seen before each of the QRS complexes and the underlying pacing rate is 60 bpm. The QRS complexes are wide (0.12 sec) and have a left bundle branch block (LBBB) morphology with a broad R wave in lead I (→) and a QS complex in lead V1 (←). There is an extremely leftward axis between –30° and –90°. However, this is not a left anterior fascicular block, since the QRS complexes are paced and hence ventricular activation is not via the normal His-Purkinje system. The QT/QTc intervals are normal (400/400 msec and 380/380 msec when corrected for the prolonged QRS complex duration). The second, fifth, and ninth QRS complexes (•) are premature, and in addition, they have a slightly wider duration (0.14 sec).

QRS complexes 3, 4, 6, 7, 8, 10, and 11 have two pacemaker stimuli, one before the P wave (*) and the second before the QRS complex (+). This represents a dual-chamber pacemaker with a lead in the right atrium and one in the right ventricle. The pacing mode seen is AV sequential pacing. The AV delay of the pacemaker is 0.22 sec.

There is a P wave (^) before the second, fifth, and ninth QRS complexes, which are premature (•). There is no pacing spike before these P waves. These are premature atrial complexes. After the P wave there is a pacemaker stimulus (↓) resulting in a wide QRS complex. Hence this is atrial- (P-wave) sensed ventricular paced or P-wave synchronous or activated right ventricular pacing. Although the QRS complex is identical in morphology to the other paced QRS complexes (*ie*, LBBB morphology), it is wider than the AV sequentially paced complexes. This is due to the fact that the premature atrial impulse reaches the AV node when it is still partially refractory. Hence conduction through the AV node is slower and ventricular activation is entirely the result of the impulse coming from the pacemaker. Thus, this QRS complex is completely captured. In contrast, the AV sequentially paced complexes, which are narrower, represent some degree of pseudofusion, as the AV delay of the pacemaker and the intrinsic PR interval are similar. The ECG shows normal DDD pacemaker function.

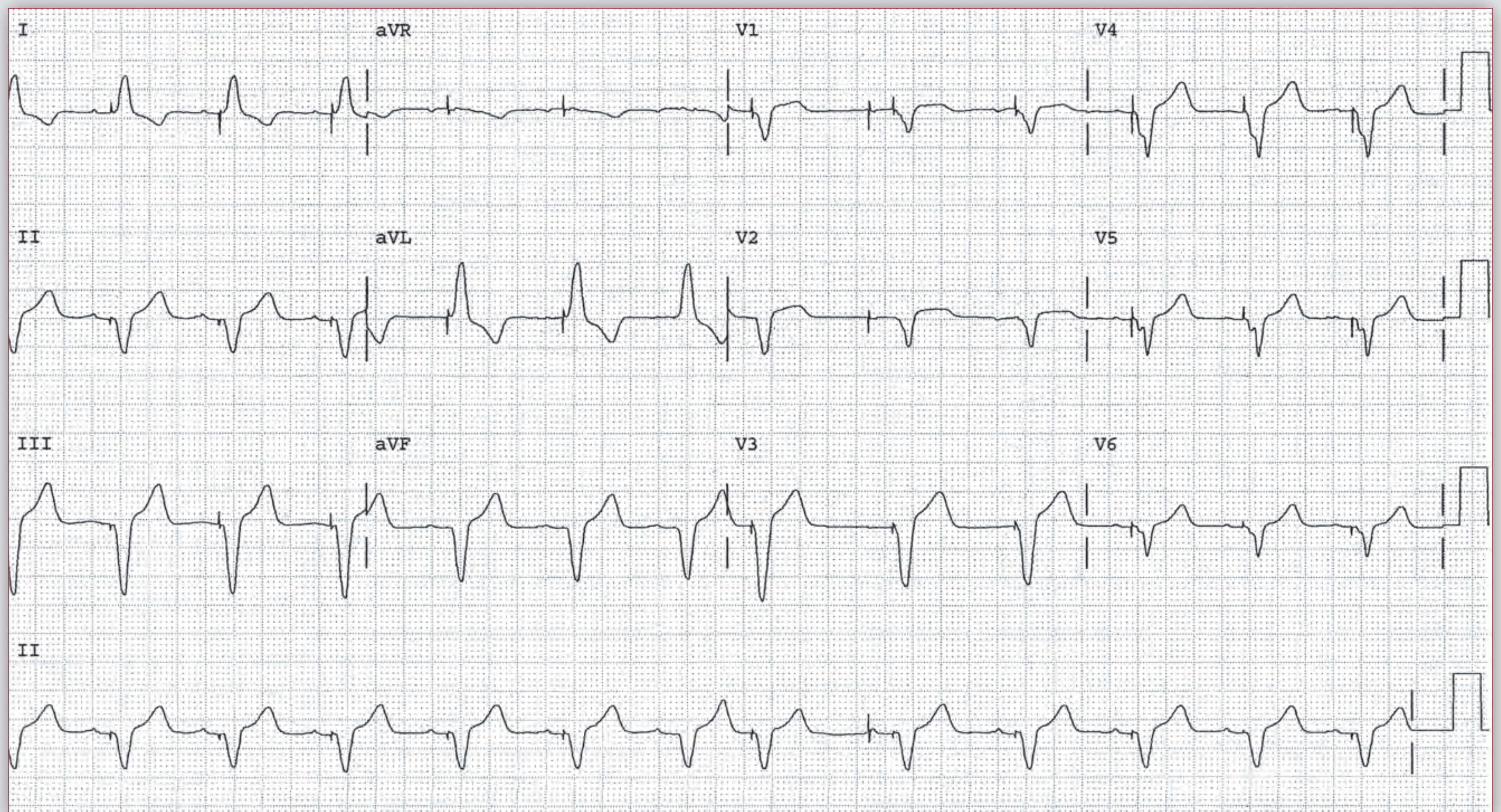
The etiology for the irregular pulse rate is premature atrial complexes that are frequently seen in the population and are of no importance. No additional therapy is necessary. Even though the patient has a pacemaker, the pacemaker will respond normally to the premature atrial impulse, resulting in a paced QRS complex. However, if the premature atrial impulse is very early, it may occur during the "blanking" period of the pacemaker, *ie*, a time when the pacemaker does not sense atrial activity. In this situation, the premature atrial complex will not result in a paced ventricular complex. This blanking period is known as the PVARP, or the post-ventricular atrial refractory period. ■

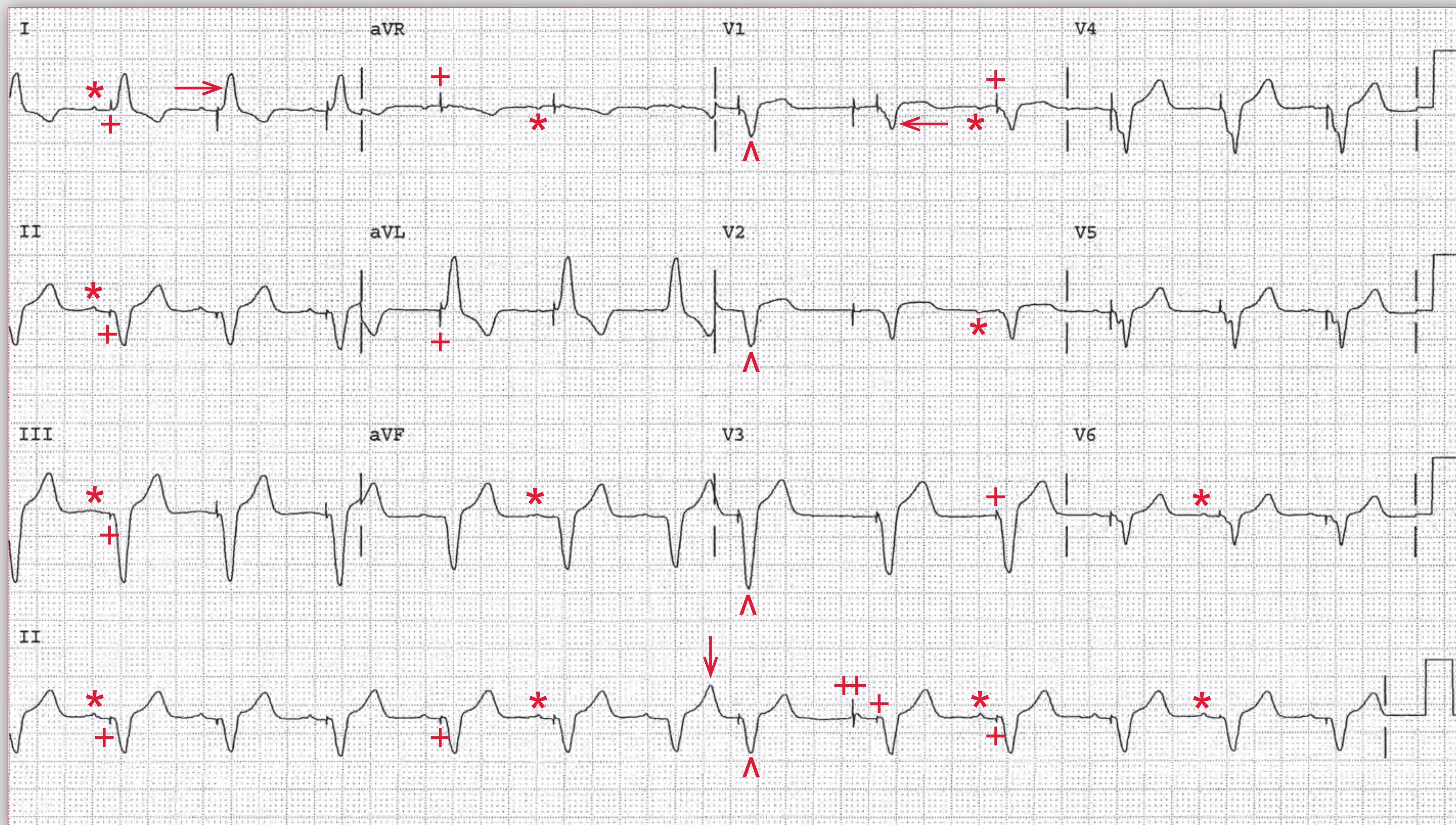
Notes

A 66-year-old woman with a history of a dilated cardiomyopathy and a previous left bundle branch block presented with a syncopal episode associated with a Mobitz type II second-degree AV block. As a result a pacemaker was inserted. When seen one year later, she is asymptomatic. An ECG was obtained.

What type of pacemaker does she have?

Is it functioning normally?





ECG 14 Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous right ventricular pacing), AV sequential pacing

There is a regular rhythm at a rate of 76 bpm; the eighth QRS complex (^), however, is premature. There is a P wave (*) seen before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is sinus rhythm. Each QRS is wide (0.16 sec) and has a left bundle branch block morphology with a broad R wave in lead I (→) and a QS complex in lead V1 (←) and an extremely leftward axis between −30° and −90°. Each of the QRS complexes is preceded by a pacemaker stimulus (+) and the pacemaker lead is in the right ventricle. Therefore, this is a dual-chamber pacemaker functioning in an atrial sensed, ventricular paced mode (P-wave synchronous ventricular pacing. There is no pacemaker stimulus seen before the seventh QRS complex, although this complex is on time and the QRS morphology is identical to the other paced QRS complexes. This means that there is intact AV conduction. The QT/QTc intervals are normal (380/430 msec and 320/360 msec when the prolonged QRS complex duration is considered).

The eighth complex is early (^) and a premature P wave can be seen; it is superimposed on the T wave, altering its morphology (↓). After the premature P wave, there is a paced QRS complex (^) followed by a pause. The complex that ends the pause is AV sequentially paced (both atrial [++] and ventricular pacing stimuli [+]) as there was no sinus P wave after the premature atrial beat. Hence the duration of the pause is equal to the lower rate limit of the pacemaker (*ie*, 76 bpm).

Hence this is a DDD (dual-chamber) pacemaker functioning in an atrial sensed ventricular paced mode (or P-wave synchronous ventricular pacing). It is functioning normally. ■

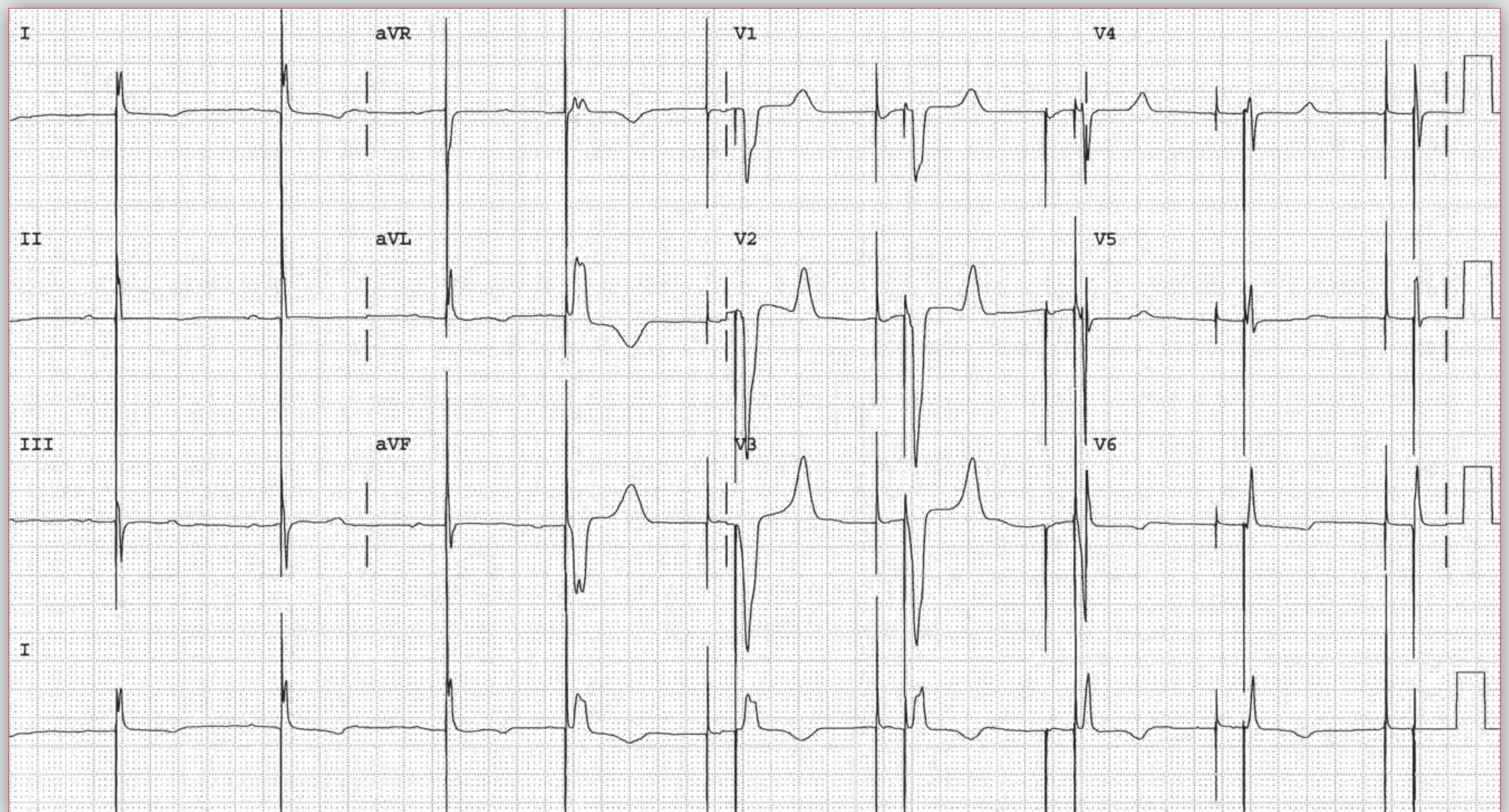
Notes

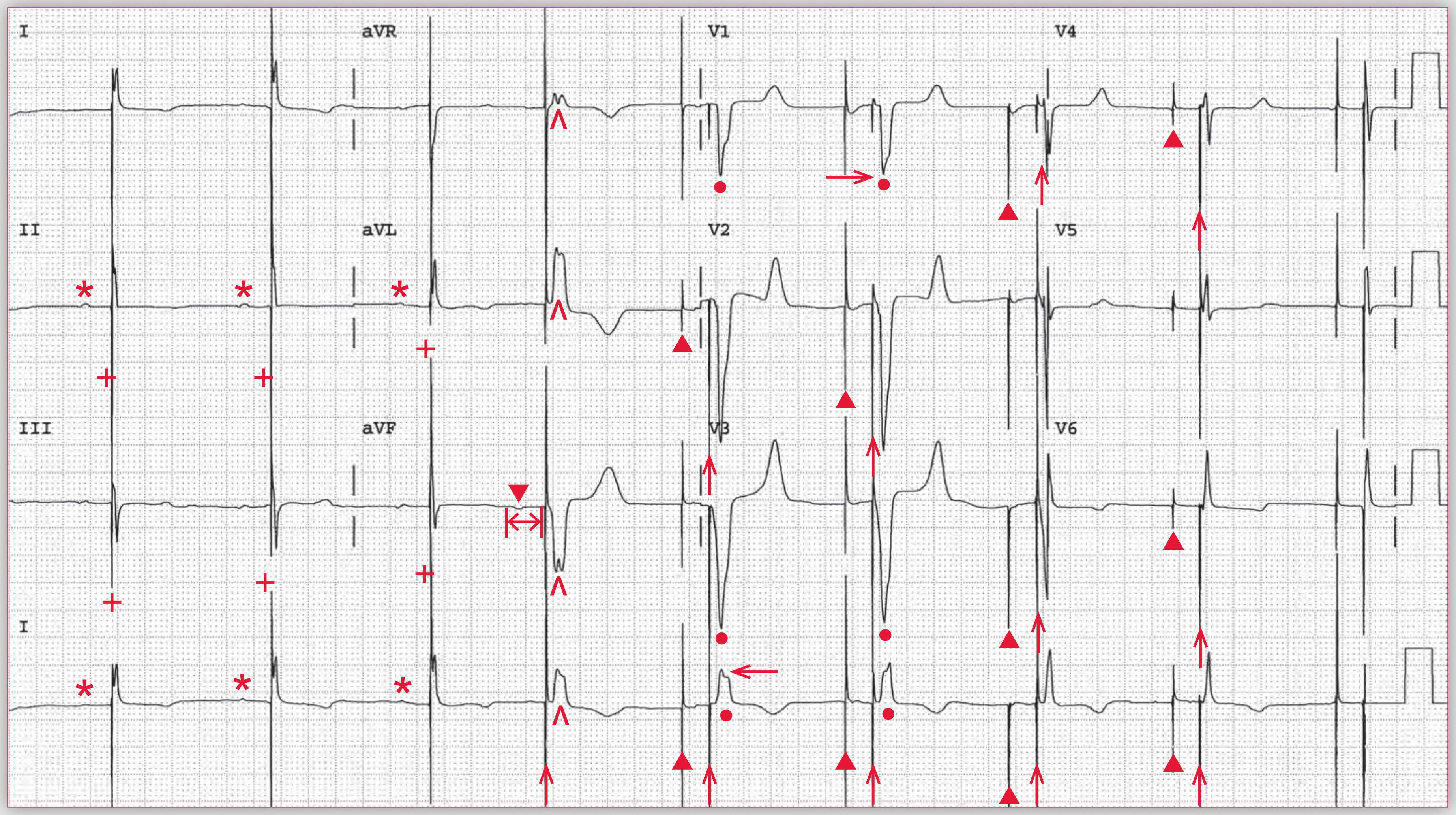
Core Case 15

A 74-year-old man is seen by his internist for a routine physical examination. He has a history of a sick sinus syndrome associated with presyncopal episodes for which he had a pacemaker inserted three years ago. At present, he has no complaints. His physical examination is normal. An ECG is obtained, and the internist becomes concerned for pacemaker malfunction and sends the patient to the emergency department.

What type of pacemaker does the patient have?

Is there evidence for pacemaker malfunction?





ECG 15 Analysis: Normal sinus rhythm, dual-chamber pacemaker, P-wave synchronous right ventricular pacing, AV sequential pacing, premature atrial complex, pseudofusion

There is a regular rhythm at a rate of 52 bpm. However, the fourth QRS complex is premature (^). The first 3 QRS complexes have a P wave (*) before them, and they are preceded by a pacemaker stimulus (+). There is a stable PR interval or AV delay (0.22 sec). Although there is a pacemaker stimulus before each of the QRS complexes, the QRS complex duration is normal (0.10 sec) and there is a normal morphology. The axis is leftward between 0° and –30° (positive QRS complex in leads I and II and negative in lead aVF). The QT/QTc intervals are normal (460/430 msec). Therefore, although there is a pacemaker stimulus, these are native QRS complexes and there is no pacemaker capture, *ie*, there is a pacemaker stimulus that occurs at the same time that there is a ventricular depolarization initiated by an impulse via the normal AV node–His-Purkinje system. As the QRS complex is via the normal AV node–His-Purkinje system, this represents pseudofusion and is due to the fact that the intrinsic AV conduction through the AV node is the same as the AV delay of the pacemaker. In this situation, ventricular activation is a result of the impulse that comes through the AV node and not one that is initiated by the pacemaker stimulus.

The fourth QRS complex (^), which is premature, is wide and abnormal. It has a left bundle branch block morphology with a broad R wave in lead I (←) and a QS complex in lead V1 (→). It is preceded by a ventricular pacemaker stimulus (↑). Hence this is a captured complex due to a stimulus originating from the right ventricle. There is a P wave (▼) before this complex and hence it is a premature atrial complex. There is an AV delay (↔) of 0.22 sec, identical to the PR interval or AV delay of the first three complexes; hence this represents an atrial sensed, ventricular paced complex (or P-wave synchronous ventricular pacing). A premature atrial complex often conducts through the AV node at a slower rate when the node is still partially refractory (due to decremental conduction seen when the rate of AV nodal stimulation increased, *ie*, in the absence of sympathetic stimulation, the faster the rate of AV nodal stimulation, the slower is conduction through the node). As a result, ventricular activation is via the pacemaker, and thus there is a completely paced complex. The fifth and sixth complexes (●) demonstrate AV sequential pacing as there is a pacing stimulus (▲) before the P wave and a pacing stimulus before the QRS complex (↑) with a stable AV delay (0.22 sec). Complexes 7, 8, and 9 also show AV sequential pacing (▲,↑), but the QRS complexes are narrow, similar to the first 3 complexes. Therefore, the ventricular complexes are pseudofused and not captured.

Hence the ECG shows a dual-chamber pacemaker with P-wave synchronous as well as AV sequential pacing. There is pseudofusion as a result of an AV delay and intrinsic PR interval that are the same. Therefore, there is no evidence for pacemaker malfunction. The pacemaker function is variable based on the intrinsic rhythm and AV conduction. ■

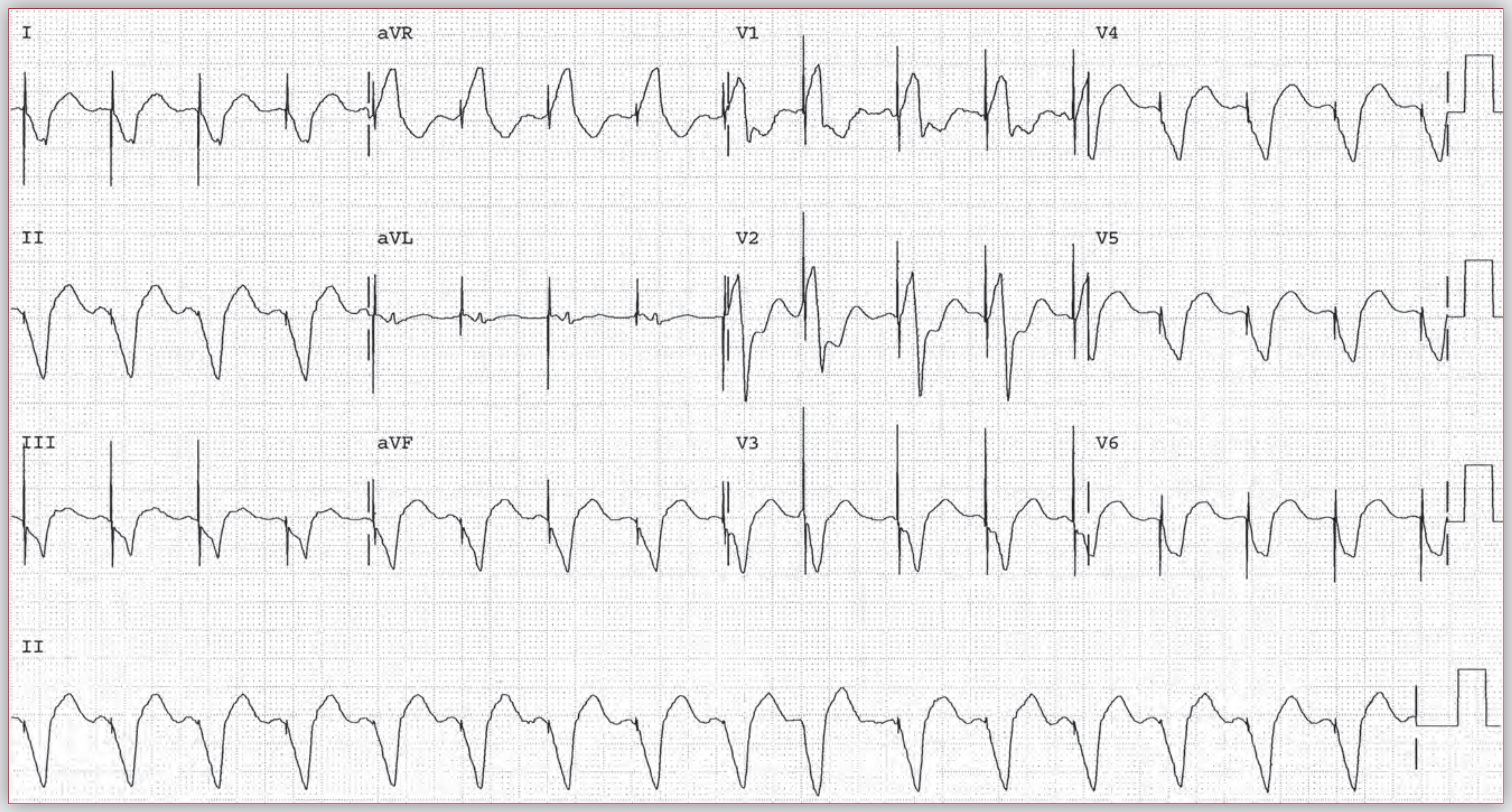
Notes

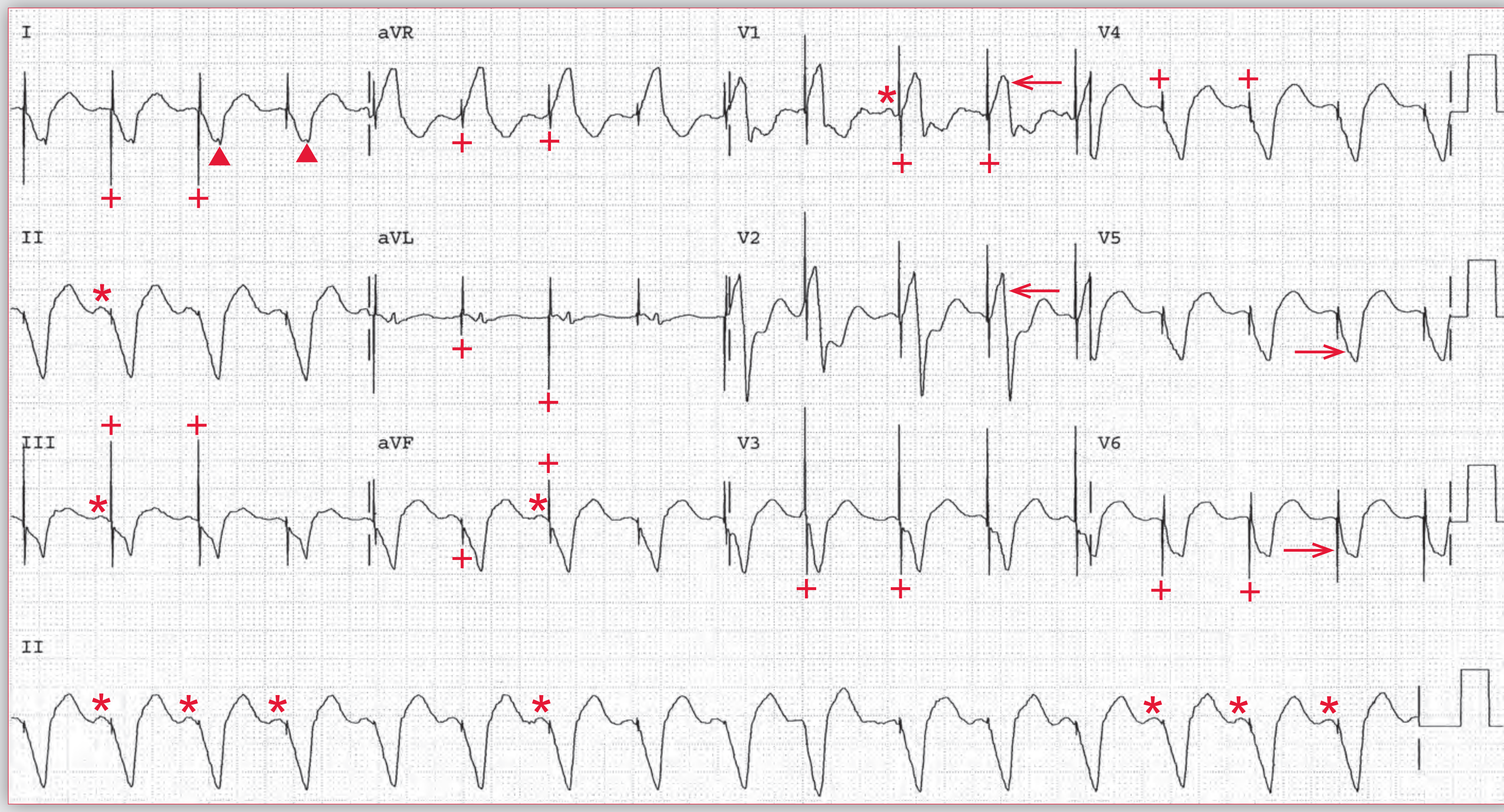
Core Case 16

A 65-year-old man has a history of a previous myocardial infarction associated with a left ventricular ejection fraction of 30%. He also has had symptoms of presyncope felt to be the result of bradycardia. He states that he had a pacemaker inserted, but is not aware of any other details. An ECG is obtained.

Does the ECG show a normally functioning pacemaker?

What type of pacemaker does this patient have?





ECG 16 Analysis: **Biventricular pacemaker, dual-chamber pacing, P-wave synchronous ventricular pacing**

There is a regular rhythm at a rate of 96 bpm. There is a P wave (*) seen before each QRS complex. The QRS complex is wide (0.20 sec) and there is a pacemaker stimulus (+) before each QRS complex; hence this is a ventricular paced beat. There is a stable PR interval (0.14 sec). Therefore, this is an atrial sensed, ventricular paced rhythm (P-wave synchronous ventricular pacing). The QT/QTc intervals are prolonged (400/505 msec) but are normal when the prolonged QRS complex duration is considered (300/380 msec).

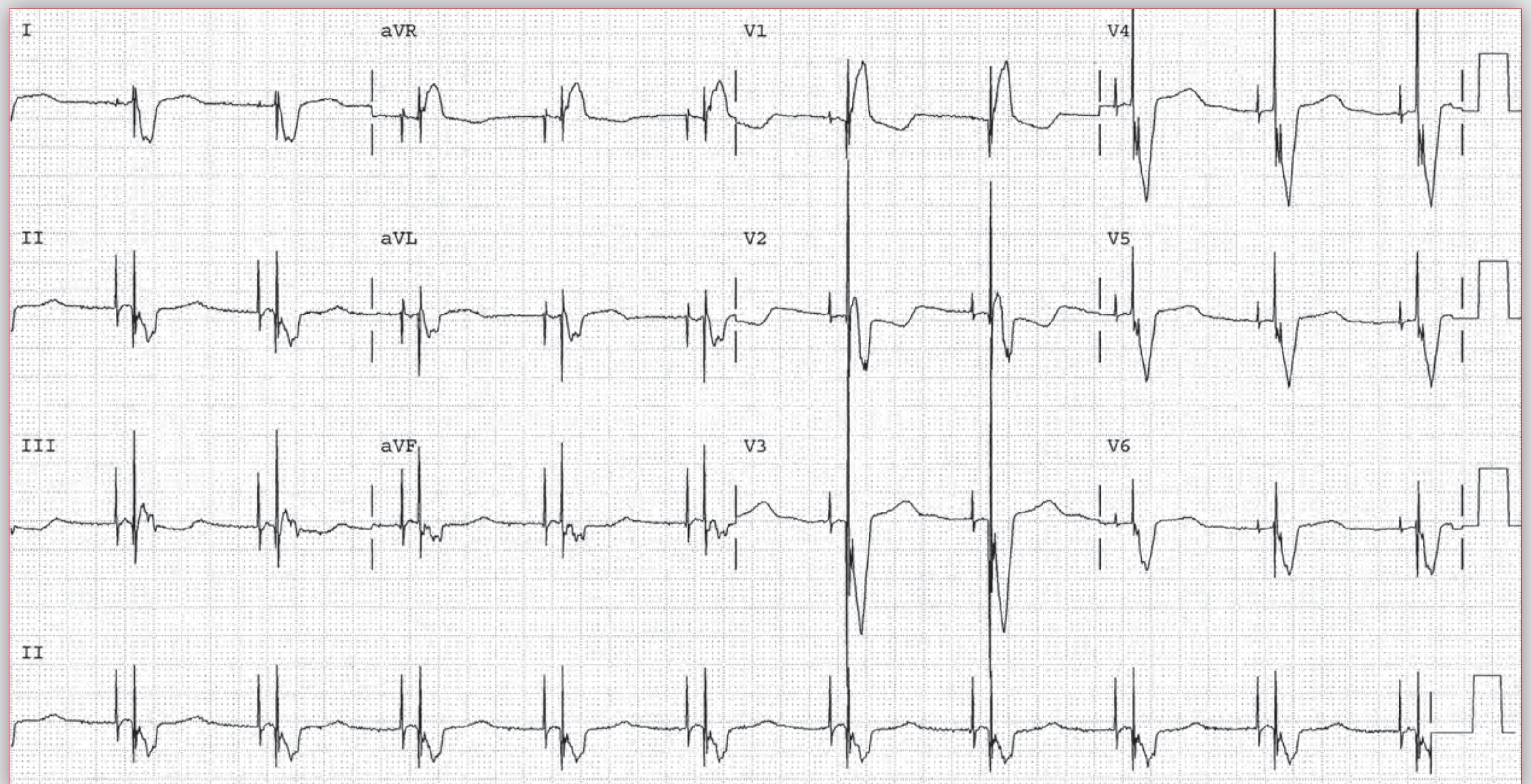
The QRS complex does not have the usual pattern resulting from a right ventricular pacemaker lead, *ie*, a left bundle branch block (LBBB) pattern with a broad R wave in leads I, and V5–V6 and a QS complex in lead V1. In contrast, leads V1–V2 has a tall and broad R wave (←) (resembling a right bundle branch block). However, this morphology may be seen when there is a right ventricular lead at the intraventricular septum. In addition, there is a QS complex in leads V5–V6 (→); however, a LBBB (due to right ventricular pacing) may have a QS complex in these leads. Therefore, the tall R wave in lead V1 and QS complex in leads V5–6 can be seen with right ventricular pacing. Importantly, there is a QS complex in lead I (▲). As a result, the axis is indeterminate between +90° and +/–180° (negative QRS complex in leads I and aVF). Lead I is a right to left bipolar lead. With normal left ventricular activation, the impulse travels from right to left, producing an R wave in this lead. With a right ventricular pacemaker, ventricular activation originates in the right ventricle and travels in a right-to-left direction. Hence lead I should always have a tall broad R wave when there is right ventricular pacing. The presence of a QS complex in lead I means that ventricular activation is in a left-to-right direction, and hence ventricular activation is initiated from the left ventricle. This is the pattern typical of biventricular (left ventricular) pacing with leads in the right ventricle as well as the coronary sinus over the left ventricle. Biventricular pacing, also known as cardiac resynchronization therapy (CRT), is used in patients with drug-refractory heart failure who have reduced left ventricular systolic function and a widened QRS complex due to a LBBB. With a LBBB, there is dyssynchronous ventricular contraction, *ie*, the septum contracts before the posterior wall of the left ventricle. With CRT, the initial activation is of the posterior wall of the left ventricle, which now contracts simultaneously with septal contraction, hence resulting in restoration of synchronous ventricle contraction. ■

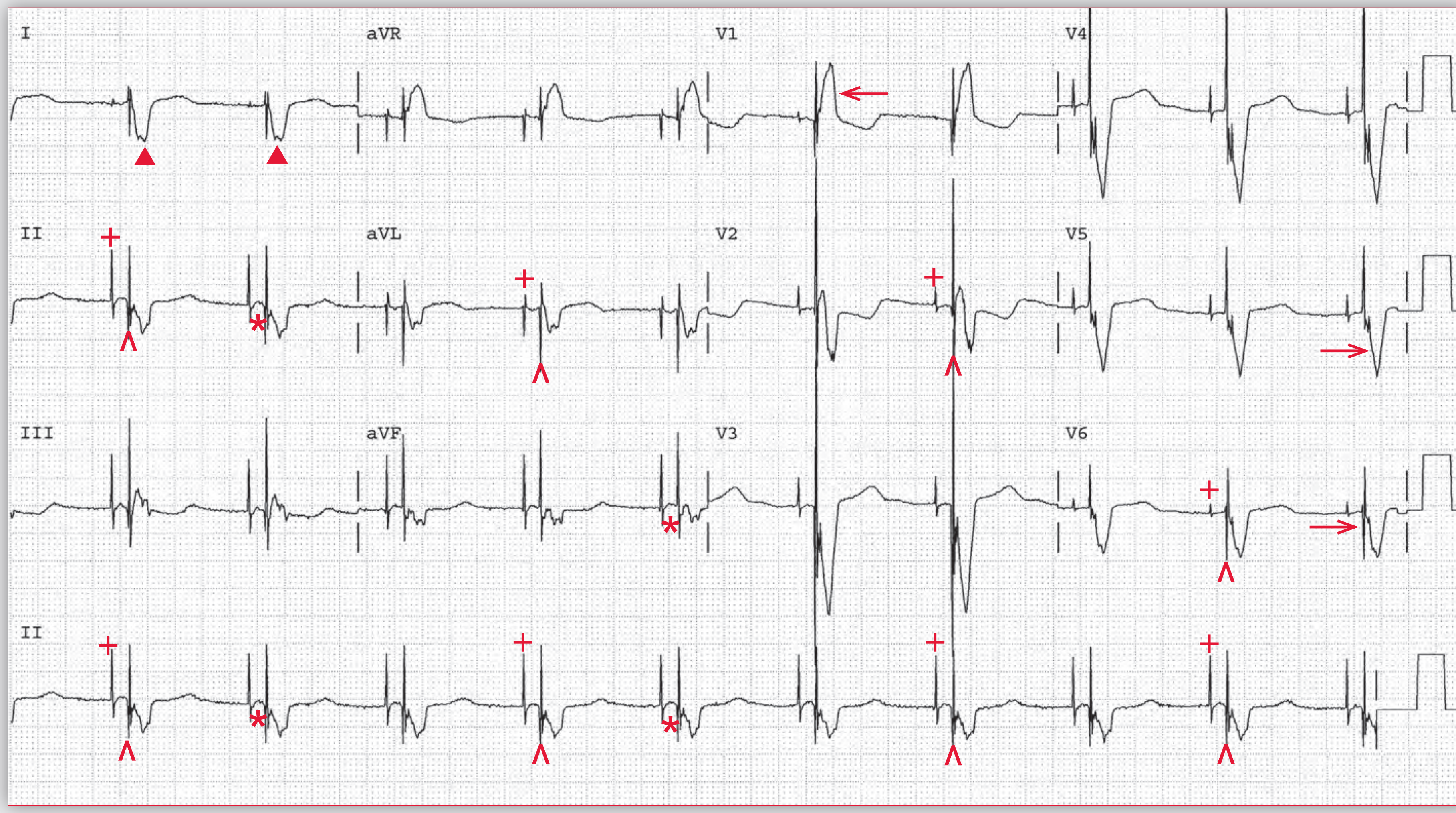
Notes

Core Case 17

A 64-year-old man with a history of a dilated cardiomyopathy and left ventricular ejection fraction (LVEF) of 25% presents to the emergency department with a two-week history of progressive shortness of breath. Upon presentation, he is noted to have a sinus tachycardia with a left bundle branch block and a sinus rate of 110 bpm, bilateral rales, a systolic murmur of mitral regurgitation and a third heart sound, neck vein distension to the earlobes, and bilateral peripheral edema to the knees. He is admitted and aggressively diuresed. In addition, he is begun on therapy with an ACE inhibitor and β-blocker. After 5 days of therapy, he loses 20 lbs. Although shortness of breath at rest is improved, he complains of some dyspnea with exertion and fatigue. As a result of a low LVEF, he has an implantable cardioverter-defibrillator (ICD) inserted for primary prevention. Prior to discharge, an ECG is obtained.

What type of pacemaker is present?





ECG 17 Analysis: Dual-chamber pacemaker, AV sequential pacemaking, biventricular pacemaker

There is a regular rhythm at a rate of 60 bpm. There are two pacemaker stimuli seen, one (+) before a P wave (*) and the second (^) before a wide QRS complex (0.16 sec). Hence this is AV sequential pacing as a result of dual-chambered pacing. The AV delay is fixed at 0.14 sec. The QT/QTc intervals are prolonged (500/500 msec) but are normal when the prolonged QRS complex duration is considered (440/440 msec).

The QRS complex does not have the usual pattern resulting from a stimulus originating from a right ventricular (RV) pacemaker electrode, which would be a typical left bundle branch block (LBBB) pattern with a broad R wave in lead I and a QS complex in leads V1 and V5–V6. The axis would usually be leftward. In contrast, lead V1 has a tall, broad R wave (←) (resembling a right bundle branch block). However, a tall, broad R wave in lead V1 may be seen with a right ventricular pacemaker lead at the RV septum. Leads V5–V6 have a QS complex (→) that is often seen with a LBBB pattern with right ventricular pacing. Importantly, lead I has a QS complex (▲). As a result, the axis is indeterminate between +90° and +/–180° (negative QRS complex in leads I and aVF). Lead I is a bipolar lead that views the impulse as it travels from the right to left arm. With a RV pacemaker, the impulse direction is from right to left, and therefore there should be a tall, broad R wave in lead I, which is a typical pattern of a LBBB. In contrast, a QS complex or an initial Q wave in lead I means that the impulse is initiated from the left and travels from the left toward the right, which indicates that this is not a RV pacemaker. Therefore, this pattern is typical for an impulse originating from the LV, *ie*, a biventricular pacemaker, which is often implanted into patients with drug-refractory heart failure and a widened QRS complex duration as a result of a LBBB, to restore synchronous contraction of the LV, *ie*, cardiac resynchronization therapy (CRT). In addition to the right atrial and right ventricular pacing lead, a pacing lead is also inserted into the coronary sinus over the LV. With a LBBB, there is dyssynchronous ventricular contraction, *ie*, the septum contracts before the posterior wall of the LV. With CRT and a LV lead, which is activated first, there is earlier activation of the posterior wall of the LV which now contracts simultaneously with septal contraction resulting in restoration of synchronous ventricle contraction.

The primary indication for the use of CRT (with or without an ICD) includes patients who have a LVEF ≤ 35%, a QRS duration greater than or equal to 0.15 sec due to a LBBB, sinus rhythm, New York Heart Association (NYHA) functional class II or III or ambulatory class IV heart failure symptoms despite optimal recommended medical therapy. CRT (with or without an ICD) may be useful and is a reasonable consideration for patients who have LVEF ≤ 35%, NYHA functional class II or III or ambulatory class IV heart failure symptoms on optimal recommended medical therapy with a QRS duration greater than or equal to 0.15 sec and a non-LBBB pattern or those with a QRS complex duration between 0.12 to 0.15 sec, atrial fibrillation that requires ventricular pacing or atrial fibrillation with reasonable rate control (either from AV nodal ablation or pharmacologic therapy). ■

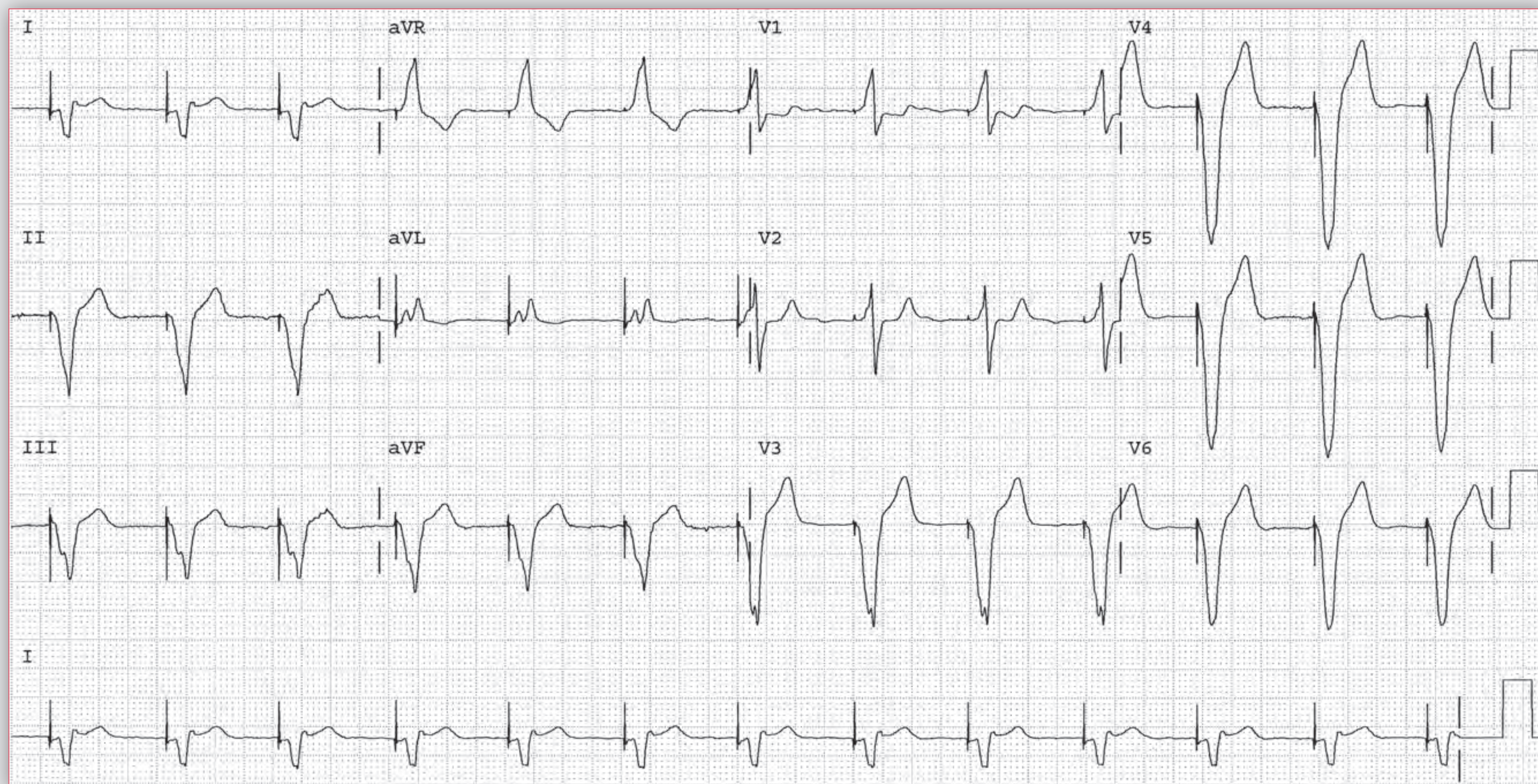
Notes

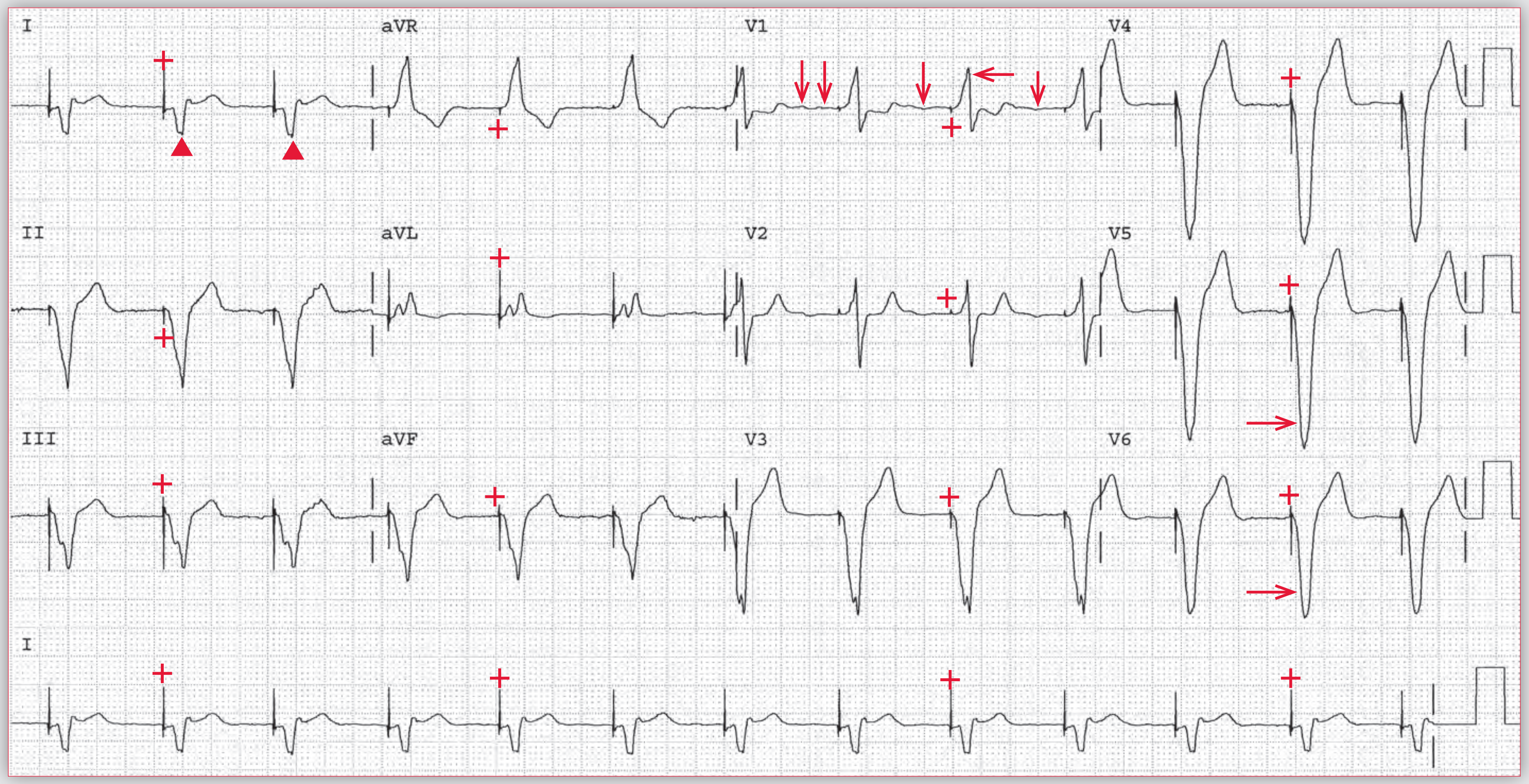
Core Case 18

A 74-year-old patient with a known nonischemic cardiomyopathy and implantable cardioverter-defibrillator (ICD), which was implanted several months ago, is brought by EMTs to the emergency department with complaints of intermittent palpitations for two weeks and a device discharge that prompted a 911 call. He noted palpitations, but no other symptoms, just prior to the device discharge.

What does the ECG show?

Does the ECG provide any information about the etiology of the palpitations and reason for the ICD discharge?





ECG 18 Analysis: Atrial fibrillation, ventricular demand pacing (VVI), biventricular pacemaker

There is a regular rhythm at a rate of 78 bpm. There is a pacemaker stimulus (+) before each QRS complex. The QRS complex duration is increased (0.16 sec), and the QT/QTc intervals are prolonged (440/500 msec) but are normal when the prolonged QRS complex is considered (380/430 msec). There is no evidence of atrial activity before or after any QRS complexes. However, there are fine irregular undulations (↓) seen in lead V1, suggesting that the underlying rhythm is atrial fibrillation. As the ventricular rhythm is regular at 78 bpm, the pacemaker is functioning in a demand ventricular pacemaker mode (VVI) and the lower rate limit is 78 bpm.

The QRS complex does not have the usual pattern resulting from a pacing lead in the right ventricle, which would be a typical left bundle branch block (LBBB) pattern with a broad R wave in lead I and a QS complex in lead V1. In contrast, lead V1 has a tall, broad R wave (←) (resembling a right bundle branch block). However, a tall, broad R wave in lead V1 may be seen with a right ventricular (RV) pacemaker lead at the RV septum. A QS morphology in leads V5–V6 (→) may be seen with a LBBB resulting from RV pacing. Importantly, lead I has a QS complex (▲). Lead I is a bipolar lead that views the impulse as it travels from the right to left arm. With a RV pacemaker, the impulse direct is from right to left and hence there should be a tall, broad R wave in lead I, which is a pattern seen with a LBBB. In contrast, a QS complex or an initial Q wave in lead I means that the impulse is traveling from the left toward the right, which indicates that this is not a RV pacemaker. Therefore, this pattern is typical for an impulse originating from the left ventricle (LV), *ie*, a biventricular pacemaker, which is often implanted into patients with drug-refractory heart failure and a widened QRS complex duration, primarily as a result of a LBBB, to restore synchronous contraction of the LV, *ie*, cardiac resynchronization therapy (CRT). In addition to the right atrial and RV pacing lead, a pacing lead is also inserted into the coronary sinus over the LV. With a LBBB, there is dyssynchronous ventricular contraction, *ie*, the septum contracts before the posterior wall of the LV. With CRT, the initial activation is of the posterior wall of the LV, which now contracts simultaneously with septal contraction, hence resulting in restoration of synchronous ventricle contraction.

The primary indication for the use of CRT (with or without an ICD) includes patients who have a LVEF ≤ 35%, a QRS duration greater than or equal to 0.15 sec due to a LBBB, sinus rhythm, New York Heart Association (NYHA) functional class II or III or ambulatory class IV heart failure symptoms despite optimal recommended medical therapy. CRT (with or without an ICD) may be useful and is a reasonable consideration for patients who have LVEF ≤ 35%, NYHA

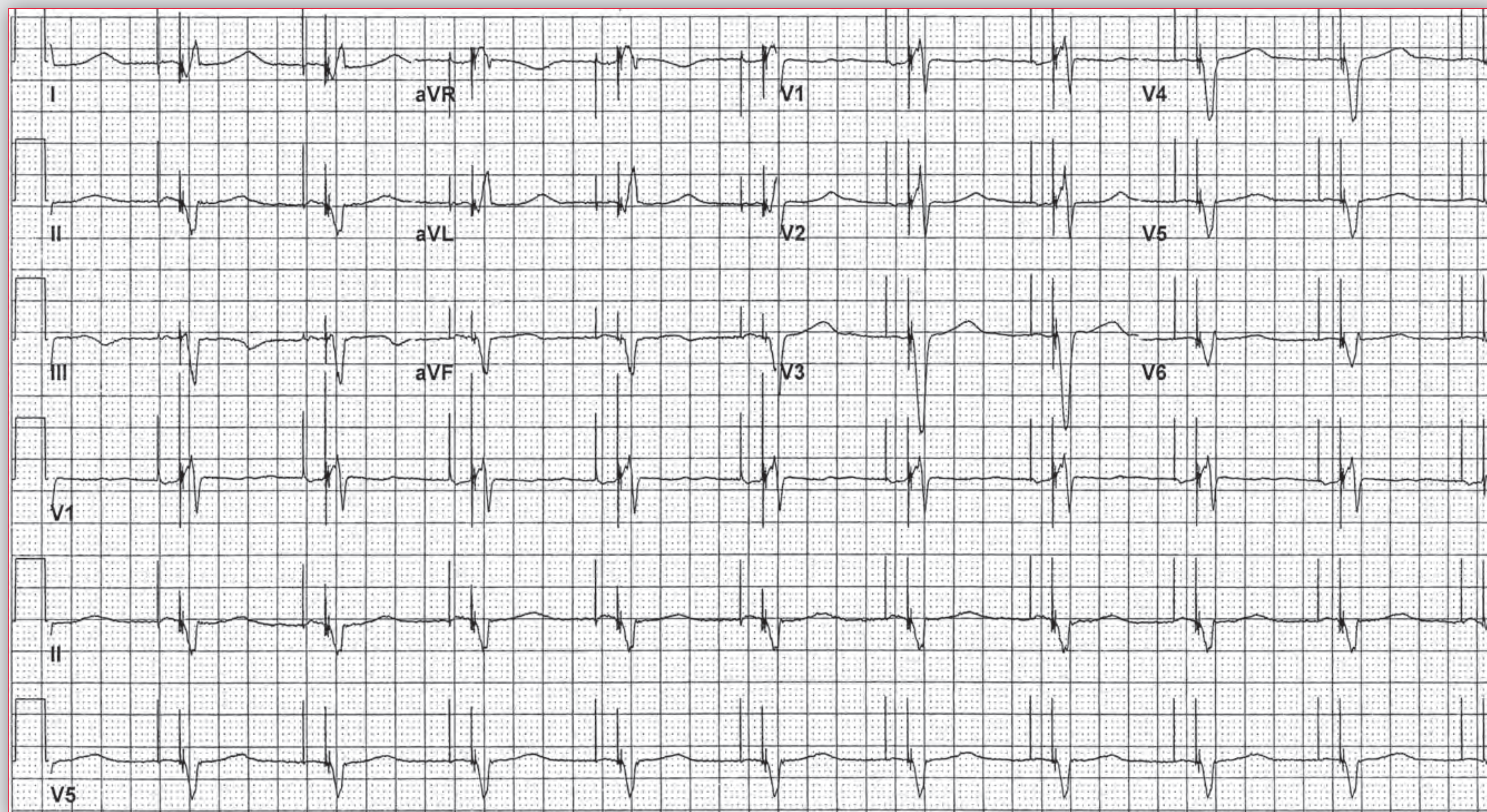
continues

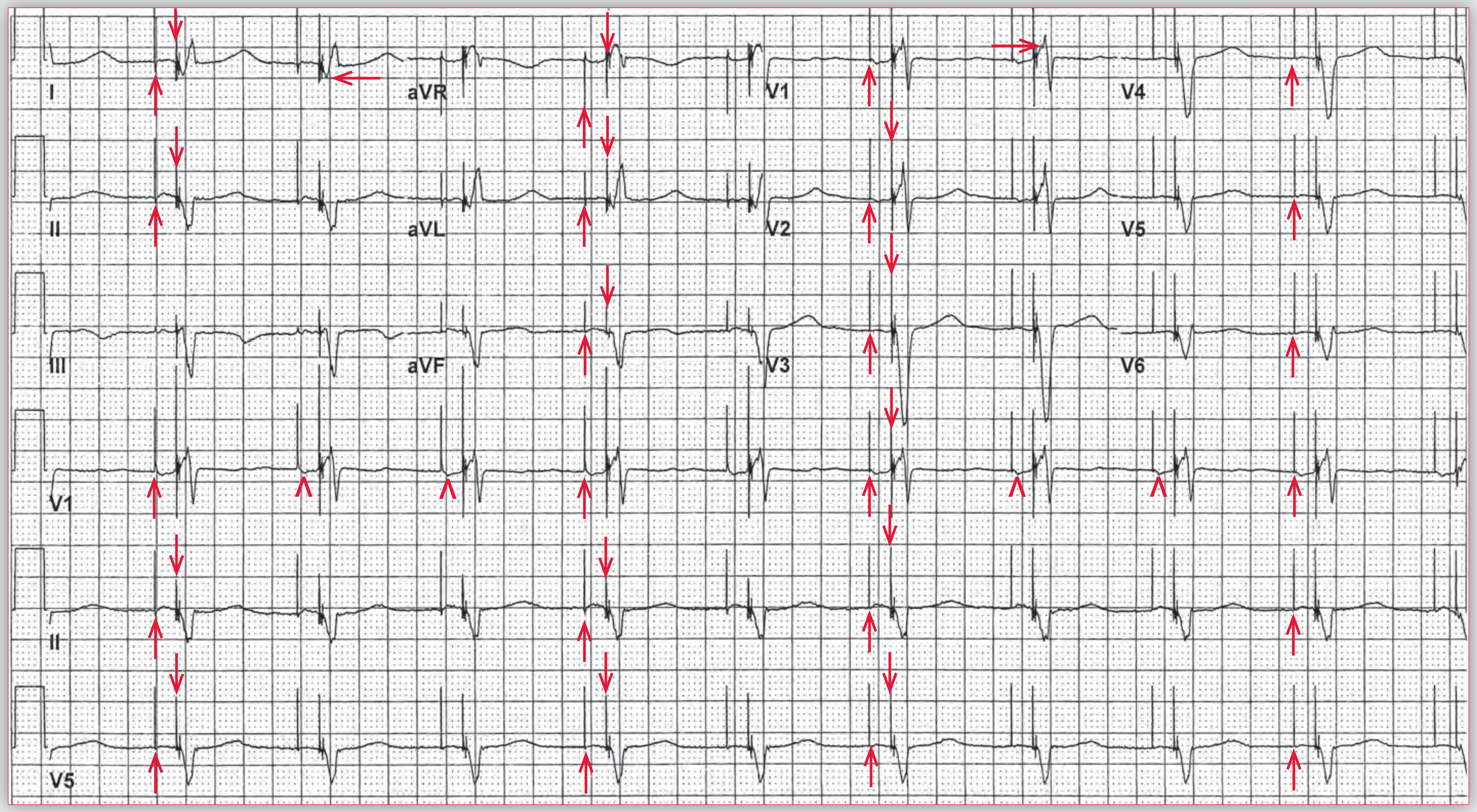
functional class II or III, or ambulatory class IV heart failure symptoms on optimal recommended medical therapy with a QRS duration greater than or equal to 0.15 sec and a non-LBBB pattern, or those with a QRS complex duration between 0.12 to 0.15 sec, atrial fibrillation that requires ventricular pacing, or atrial fibrillation with reasonable rate control (either from AV nodal ablation or pharmacologic therapy).

The finding of atrial fibrillation as an underlying rhythm suggests that the etiology of the palpitations might be atrial fibrillation with a rapid ventricular response. It is possible that this is also the etiology for the ICD discharge that was preceded by palpitations but no other symptoms. Interrogation of the ICD would provide important information about the actual cause of the palpitations and the reason for device discharge. ■

A 65-year-old man presents to his primary care physician for a first visit. He has a history of heart failure. One year before, he had a pacemaker inserted for bradycardia, although he does not know any further details. An ECG is obtained.

What type of pacemaker is present?





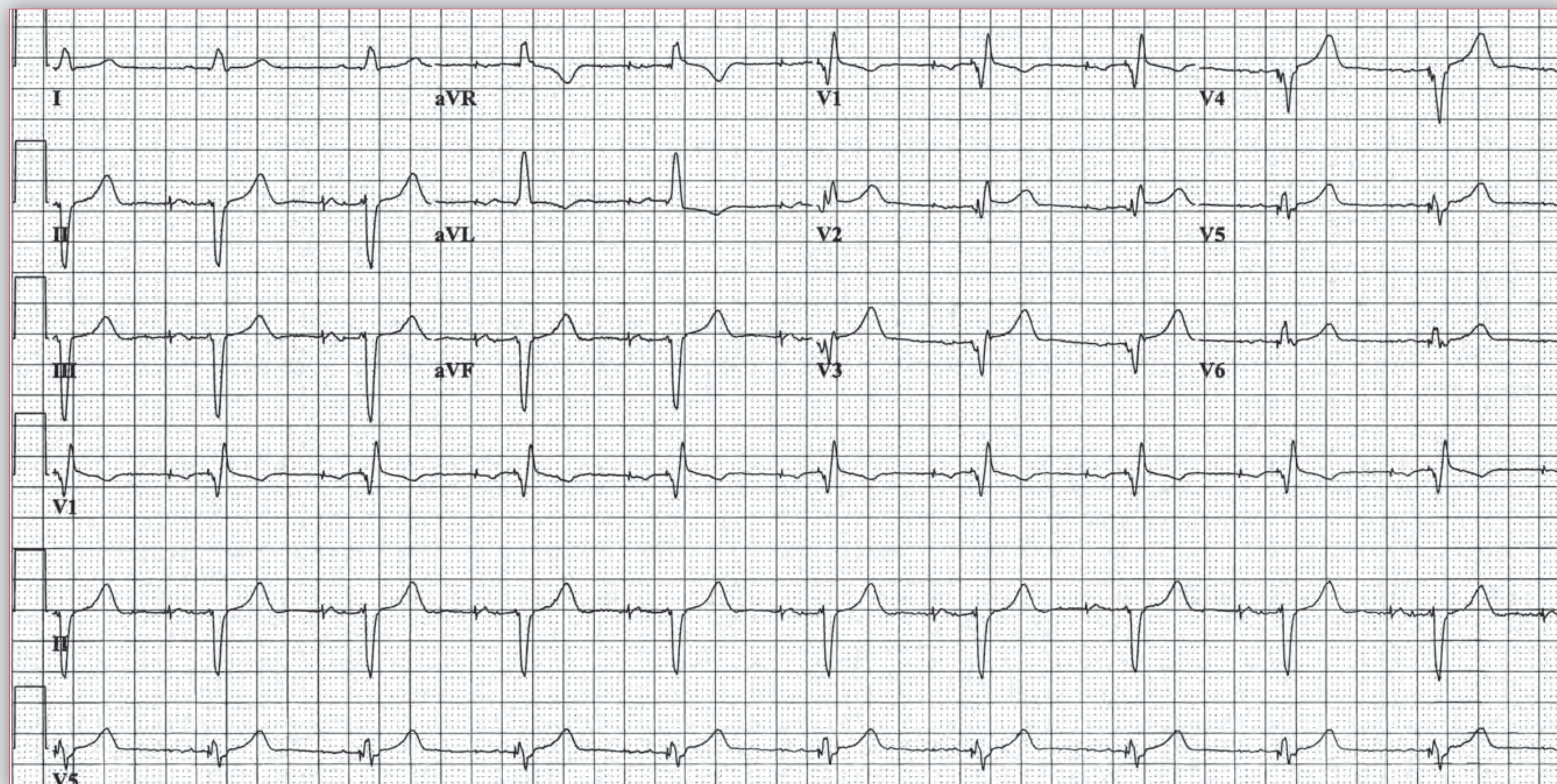
ECG 19 Analysis: Dual-chamber pacemaker, AV sequential pacing, biventricular pacing

There is a regular rhythm at a rate of 60 bpm. Two pacing stimuli are present. The first one (↑) is before the P wave (^) while the second one (↓) is before the QRS complex. Hence, there is a dual-chamber pacemaker that is functioning in an AV sequential mode. The AV delay is 0.16 sec. The QRS complex is wide (0.14 sec) and the morphology is not typical for a right (RBBB) or left bundle branch block (LBBB). Therefore, the QRS complex morphology is not typical of a right ventricular (RV) pacemaker. There is a tall R wave in V1 (→), indicating that the impulse is traveling in a left to right direction, which is not seen with a pacing from the RV as there should be in a LBBB pattern or a QS complex in V1. However, this tall R wave may be seen with a RV lead at the septum. In addition, there is a QS complex in leads V5–V6 indicating impulse conduction going from left to right; on occasion, this may be seen with a LBBB. More important, the initial waveform in lead I is a Q wave (←), indicating that the initial impulse direction is from left to right, which is not seen in a LBBB or with RV pacing. The presence of an initial Q wave in lead I indicates that there is LV pacing or biventricular pacing. Also seen are two ventricular pacing stimuli, most apparent in leads II, aVF, and V4–V6. This is due to outputs from both the LV and RV electrodes. ■

Core Case 20

A 71-year-old woman with a history of hypertension and presyncopal episodes felt to be the result of sick sinus syndrome, for which a pacemaker was implanted, is seen by her internist for a routine followup visit. She does not

ECG 20A



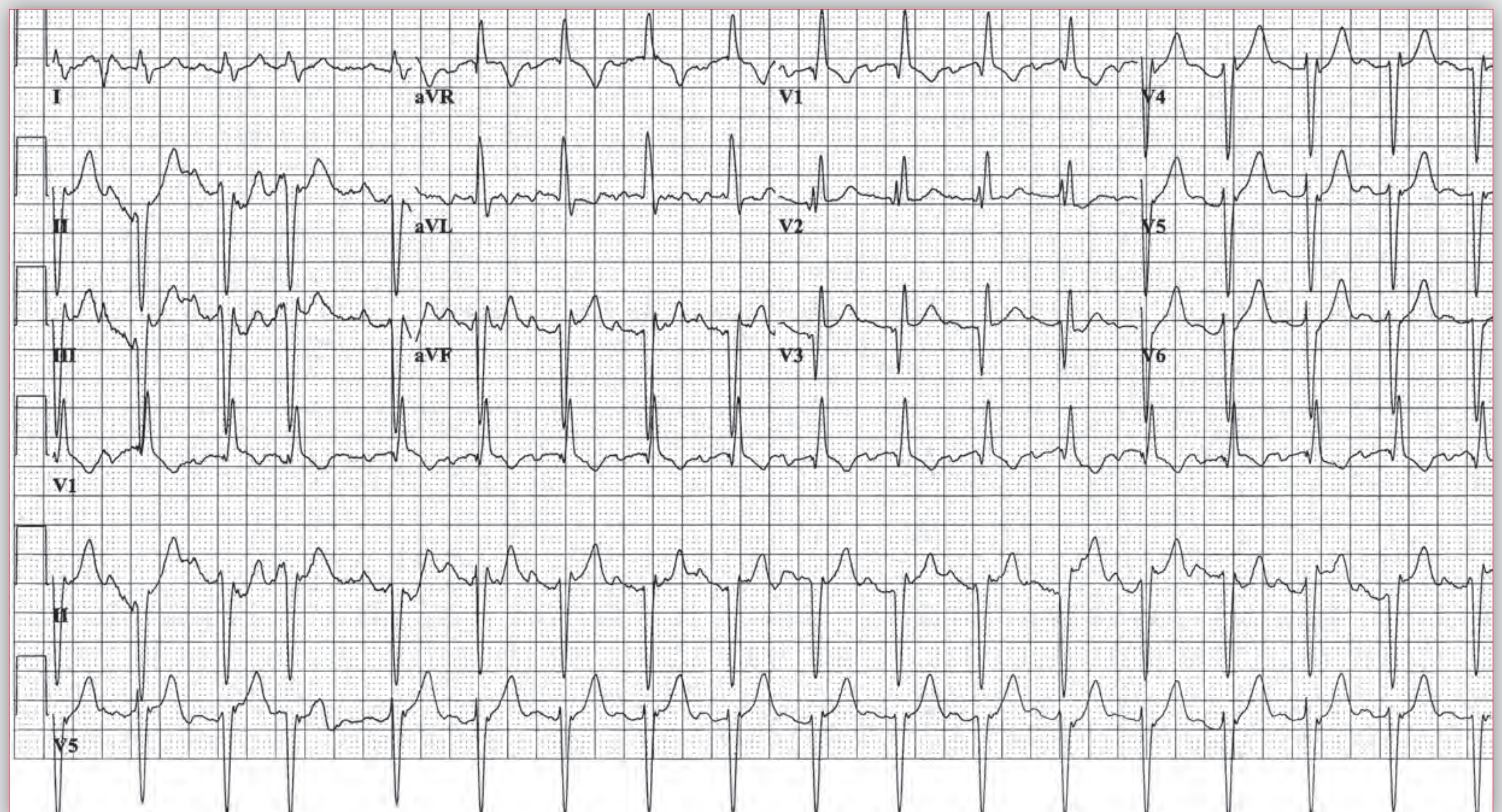
have any complaints. A routine ECG is obtained. The ECG (ECG 20A) is compared to a previous ECG (ECG 20B), and a change in the QRS complex is noted. The patient is urgently referred to her cardiologist for further evaluation of a pacemaker problem.

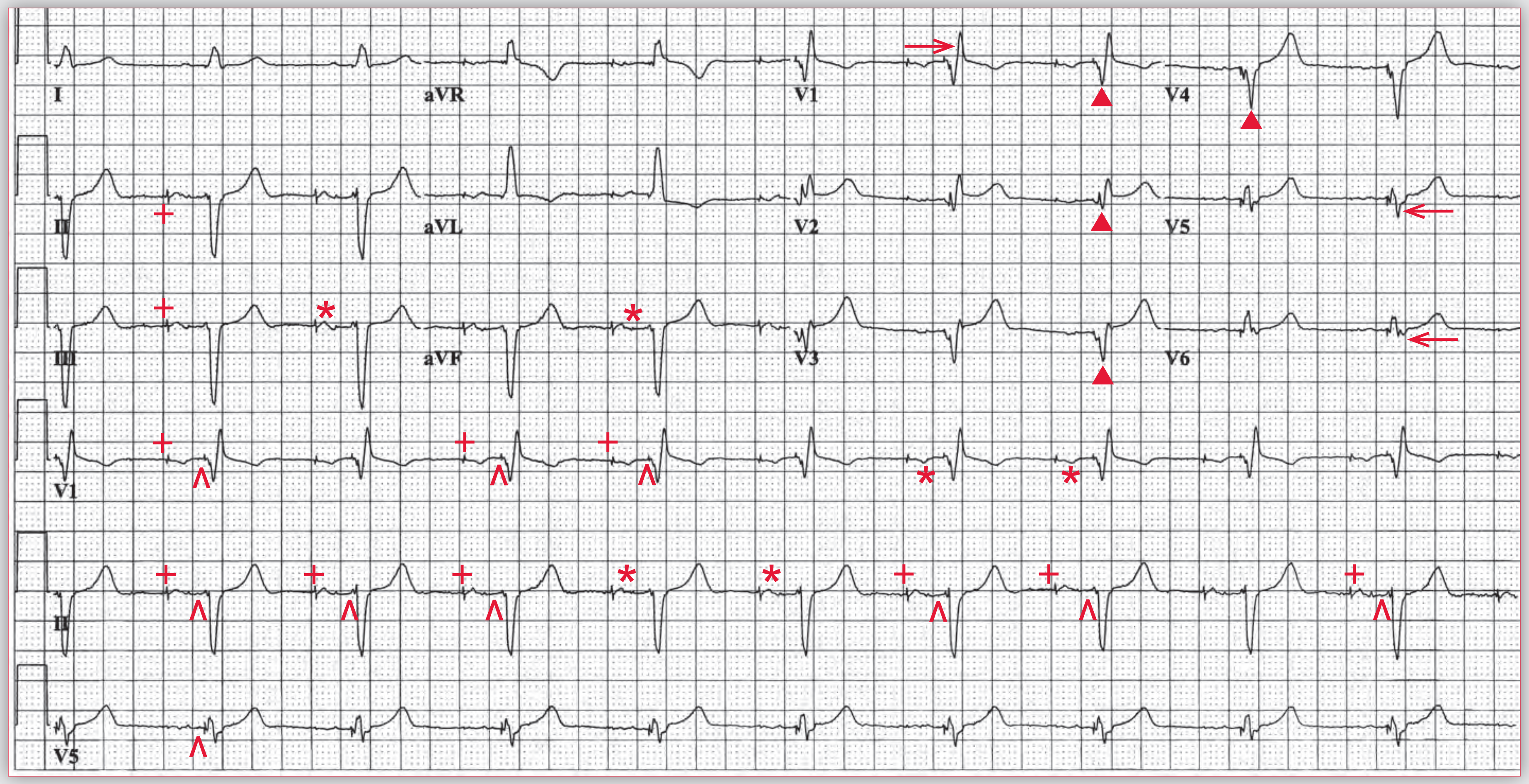
What does the ECG show?

Is there evidence for pacemaker malfunction?

Is further therapy necessary?

ECG 20B





ECG 20A Analysis: Dual-chamber pacemaker, AV sequential pacing, pseudofusion, right bundle branch block, left anterior fascicular block, old anterior wall myocardial infarction

ECG 20A shows there is a regular rhythm at a rate of 60 bpm. There are two pacemaker stimuli seen. The first (+) is before a P wave (*) and the second (^) is before a QRS complex that has an increased duration (0.12 sec). The AV delay is 0.24 sec. This is a dual-chamber pacemaker functioning in an AV sequential pacing mode. However, the QRS complexes do not have a morphology typical of a right ventricular (RV) pacemaker, which would be a left bundle branch block (LBBB) pattern (broad R wave in leads I and V5–V6 and QS complex in lead V1), or a biventricular pacemaker, which would be a QS complex in leads I and V5–V6 and a tall, broad R wave in V1. The QRS complex in lead V1 has a qR morphology (→) and in V5–V6 there is a small broad terminal S wave (←). Hence there is a typical right bundle branch block (RBBB) morphology. In addition, there is a prominent Q wave (▲) in leads V1–V4, characteristic of an old anterior wall myocardial infarction. The axis is extremely leftward between −30° and −90° (positive in lead I and negative in leads II and aVF with a rS morphology), diagnostic of a left anterior fascicular block (LAFB). The QT/QTc intervals are normal (420/420 msec and 400/400 msec when the prolonged QRS complex duration is considered). Hence the morphology suggests that this is not a paced QRS complex, but rather it is a native QRS complex. This would mean that the ventricular pacemaker stimulus is not capturing the ventricle.

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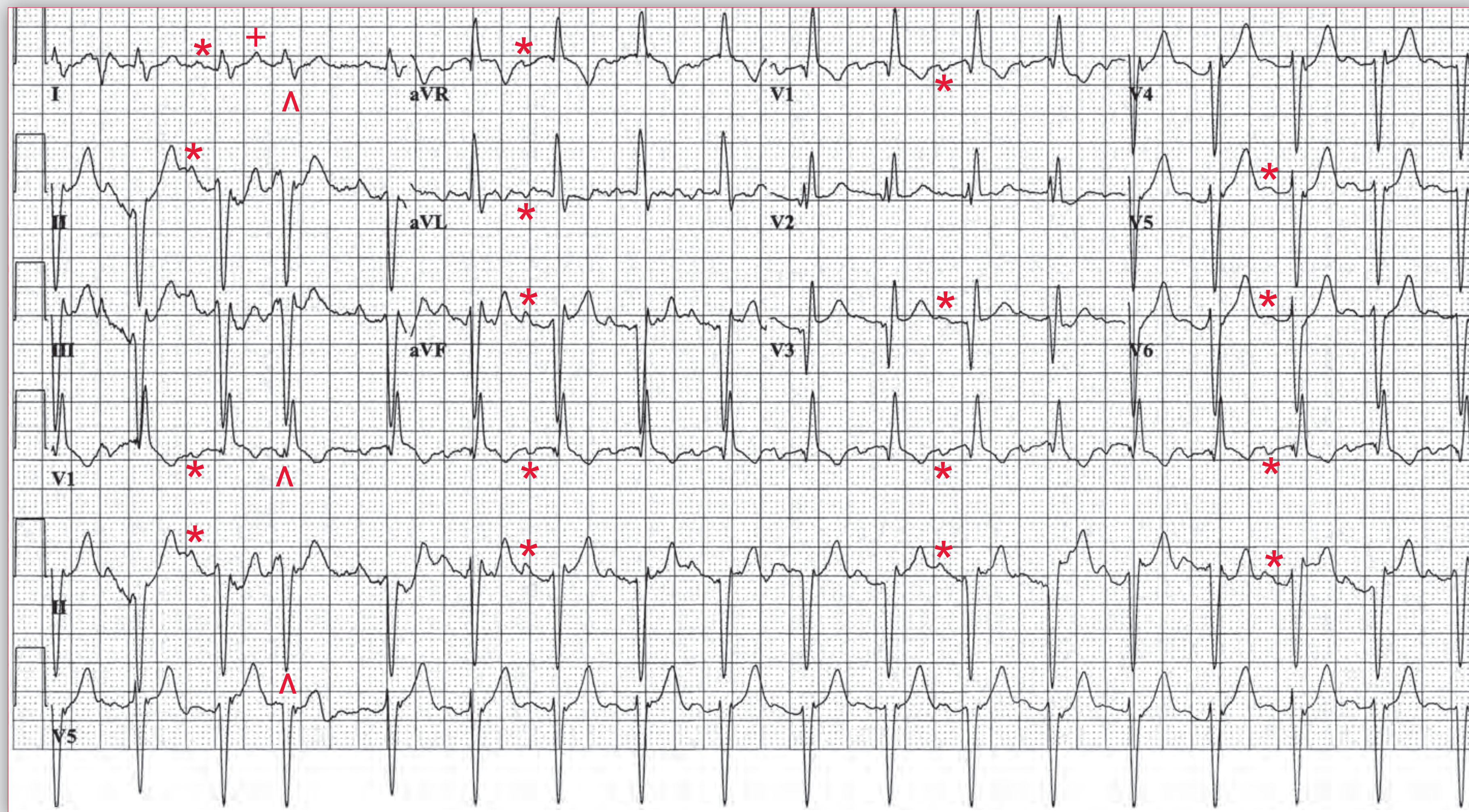


ECG 20B Analysis: **Sinus tachycardia, first-degree AV block, premature atrial complex, right bundle branch block, left anterior fascicular block, old anterior wall myocardial infarction**

This can be confirmed by looking at **ECG 20B**, which is from the same patient. This shows a regular rhythm at a rate of 104 bpm. No pacemaker stimuli are seen. There is a P wave (*) before each QRS complex with a stable PR interval of 0.24 sec, which is the same as the AV delay of the pacemaker seen in ECG 20A. The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is sinus tachycardia with a first-degree AV block. The fourth QRS complex (^) is premature. It has a morphology that is identical to the sinus complexes. A possible P wave (+) preceding the QRS complex can be seen in lead I; the T wave morphology is slightly different from that of the sinus complex, suggesting a superimposed P wave. Thus this is a premature atrial complex.

The QRS complex duration is increased (0.12 sec), and it has the same morphology as the QRS in ECG 20A, *ie*, a typical RBBB pattern, LAFB, and anterior wall myocardial infarction. The QT/QTc intervals are the same. Hence the QRS complexes in ECG 20A are native QRS complexes and the pacemaker is not capturing the ventricle. This is termed pseudofusion that is not pacemaker malfunction (*ie*, failure to capture), as the failure of ventricular capture is due to the fact that the intrinsic AV conduction through the AV node is the same as the AV delay of the pacemaker. In this situation, there is ventricular activation resulting from the impulse traveling through the AV node–His-Purkinje system rather than activation resulting from the pacemaker stimulus. As there is intact AV conduction, the pacemaker stimulus is ineffective (does not result in ventricular activation) and not necessary. Therefore, if this is a standard RV pacemaker, this is best treated with increasing the AV delay of the pacemaker to avoid unnecessary pacemaker output. However, if the patient has a biventricular pacemaker, it would be important to shorten the AV delay of the pacemaker so that it continuously captures as continuous left ventricular pacing is necessary to derive benefit from biventricular pacing. ■

Notes

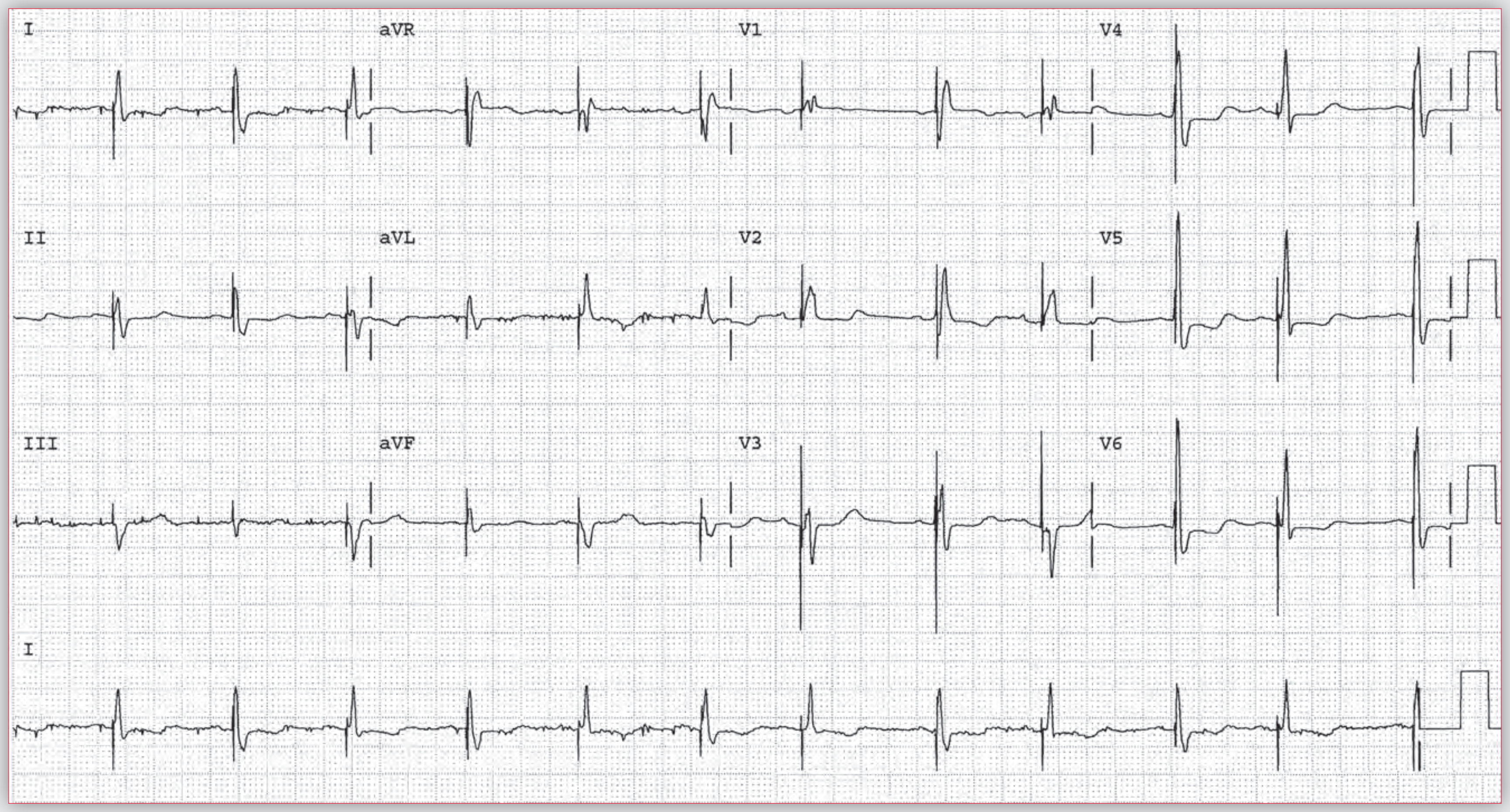
Core Case 21

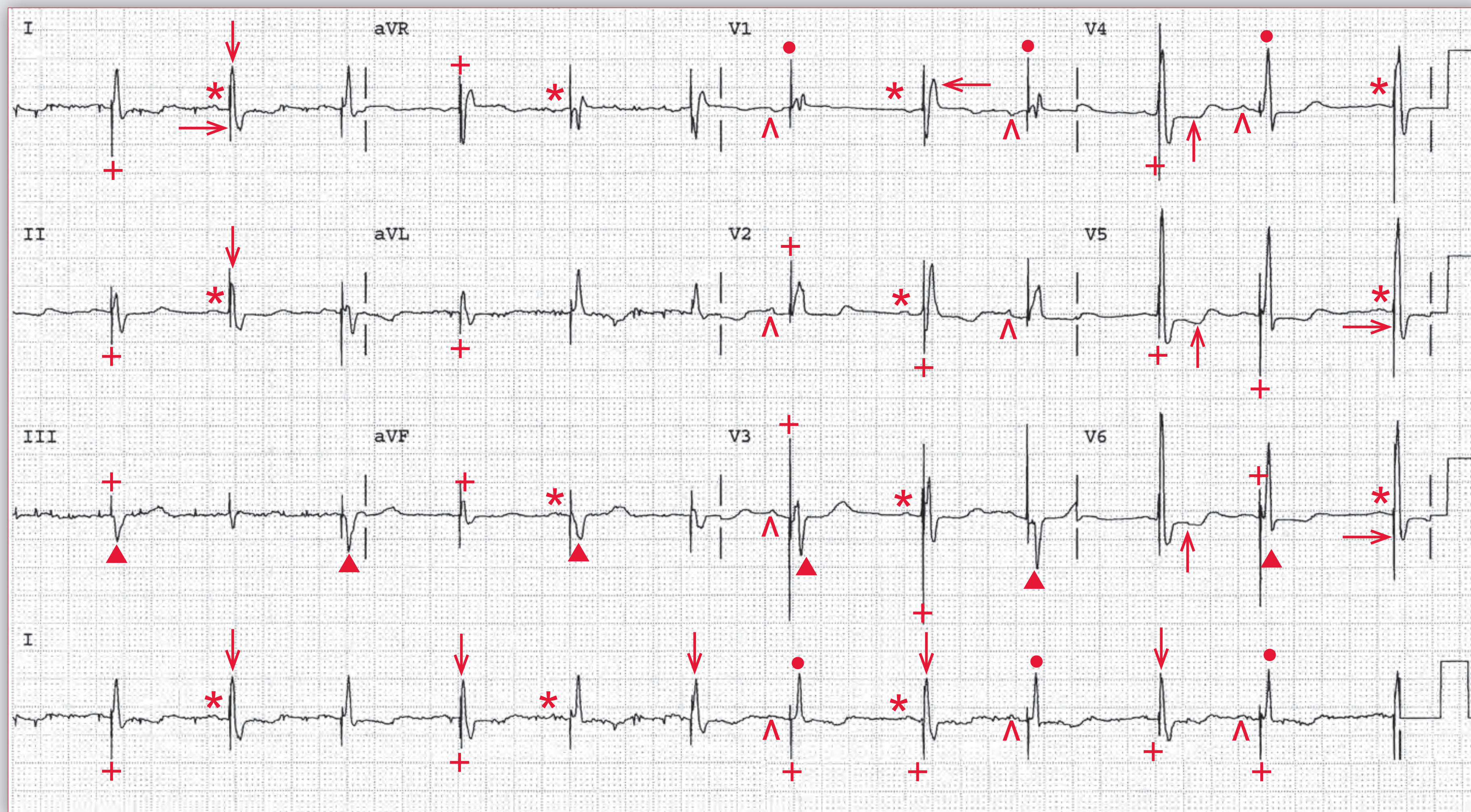
A 81-year-old man with a history of presyncopal episodes felt to be a result of sinus bradycardia presents to the emergency department with multiple episodes of exertional chest discomfort. He has an episode of chest discomfort while in the emergency department and an ECG is obtained. It is noted that there is a pacemaker present, and hence it is felt that the ECG cannot be used for establishing the presence of ischemic changes, as it is in uninterpretable.

What does the ECG show?

Is pacemaker function normal?

Can the presence of ischemic changes be assessed on this ECG?





ECG 21 Analysis: Sinus rhythm, premature atrial complexes, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous ventricular pacing), pseudofusion, ST-T wave changes

The rhythm is basically at a rate of 72 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence there is a normal sinus rhythm. However, complexes 7, 9, and 11 (•) are slightly premature and they have P wave before them (^), but there is a subtle difference in the P-wave morphology. These are premature atrial complexes (PACs).

Prior to each QRS complex (including the PACs) there is pacing stimulus (+). Therefore, this is a dual-chamber pacemaker with P-wave synchronous ventricular pacing (atrial sensed, ventricular paced). The QRS complex duration is prolonged (0.12 sec). However, the morphology is not typical for either a right ventricular pacemaker (which should have a left bundle branch block [LBBB] morphology) or a biventricular pacemaker (which has a QS complex in leads I and V5–V6 and a tall R wave in lead V1). Therefore, the ventricular pacemaker stimulus is not capturing the ventricle. Instead the QRS complex is a native complex, the result of conduction via the AV node–His-Purkinje system. This represents pseudofusion that occurs when the intrinsic AV conduction time is the same as the AV delay of the pacemaker. In this situation there is normal ventricular activation via the AV node that does not result in suppression of the ventricular output as the ventricular output and stimulation is simultaneously occurring.

Noted, however, are differences in the QRS morphology. This is the result of different degrees of fusion between the pacemaker stimulus and conduction via the normal AV node–His-Purkinje system, resulting from slight changes in AV nodal conduction time. It can be seen that every other QRS complex (↓) has a morphology that is typical of a right bundle branch block, with an RSR′ in V1 (←) and a broad S wave in leads I and V5–V6 (→). The alternating QRS complexes (*ie*, the PACs) have a different morphology (▲); they do not have a terminal broad S wave but rather have a slurred and broad upstroke, especially obvious in leads V4–V6, and also a leftward axis. This QRS complex is more fused, *ie*, more of ventricular myocardium is activated via the pacemaker and less as a result of the pacemaker stimulus. This is due to the fact that the premature atrial impulse, arriving at the AV node slightly early, is conducted more slowly through the node as a result of decrimental conduction. Hence more of the ventricular myocardium is innervated via the pacemaker stimulus.

The QRS complexes with the right bundle branch block complex are not paced but are the native complexes. They have a normal axis between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are prolonged (420/470 msec) but are normal when the prolonged QRS complex duration is considered (400/440 msec). As these complexes are not paced, the QRS complex and ST-T wave abnormalities can be interpreted and hence abnormalities effecting the left ventricle can be diagnosed. This includes the presence of myocardial ischemia. Indeed, there are 1-mm horizontal ST-segment depressions noted in leads V4–V6 (↓) which are characteristic of active ischemia, particularly given the clinical history of chest discomfort. ■

Notes

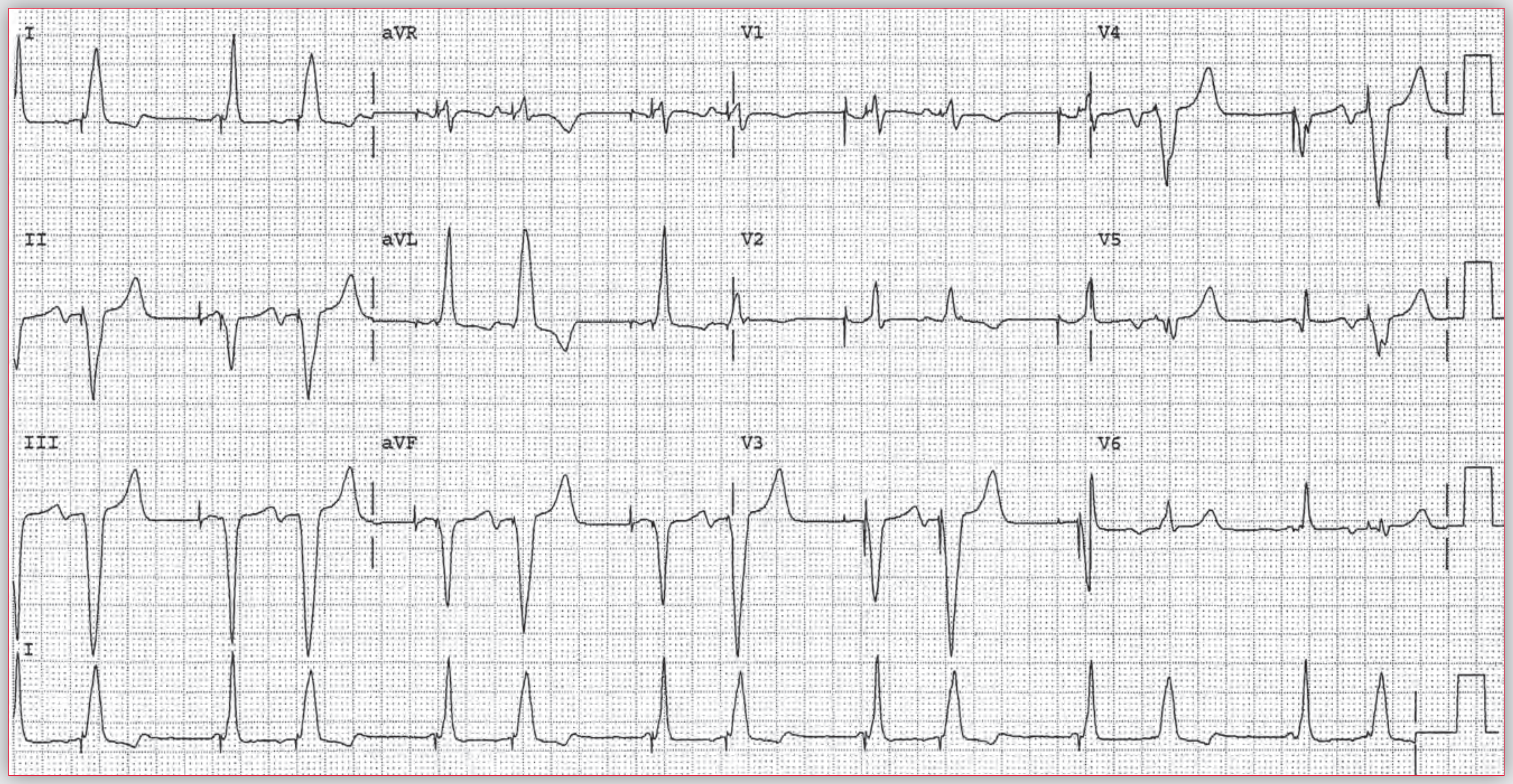
Core Case 22

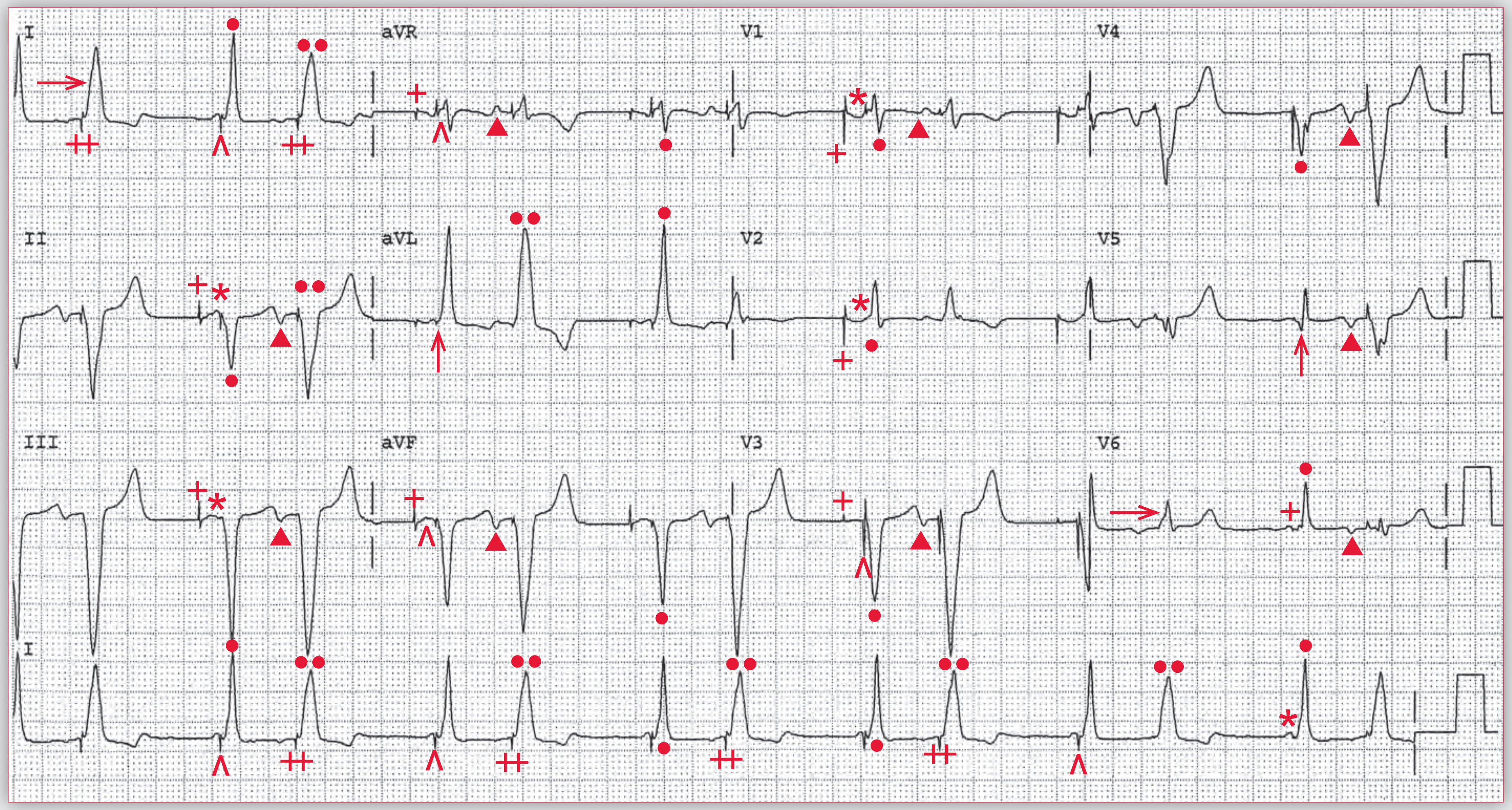
A 65-year-old woman presents to her internist with complaints of an irregular pulse, which she has noted over the past two weeks. She has been taking her pulse on a regular basis since an episode of paroxysmal atrial fibrillation several years before. She states that her pulse had been regular until two weeks ago. Although she does not have any symptoms, she is concerned about recurrent atrial fibrillation. She also mentions that she had a pacemaker inserted at the time of her atrial fibrillation, although she is not aware of the details. An ECG is obtained. Her internist is concerned about the ECG and hence he refers the patient to a cardiologist for further evaluation.

Is atrial fibrillation present?

What type of pacemaker does this woman have?

Is pacemaker function normal?





ECG 22 Analysis: Dual-chamber pacemaker, AV sequential pacing, atrial bigeminy demonstrating P-wave synchronous ventricular pacing, pseudofusion

The rhythm is irregular, but there is a pattern of long and short intervals. Hence the rhythm is regularly irregular. The average rate is 78 bpm. The QRS complex after the pause (●) has two pacemaker stimuli. The first (+) is before the P wave (*) and the second (^) before the QRS complex. This is a dual-chamber pacemaker functioning in an AV sequential mode. The AV delay is 0.16 sec and the QRS complex duration is prolonged (0.12 sec). However, the QRS complex does not have features of a typical left bundle branch block (LBBB), which would be seen with a right ventricular (RV) pacemaker. For example, there is an initial septal Q wave noted in leads aVL and V5 (↑). Septal forces or Q waves are not seen with a LBBB as this waveform originates from a septal or medial branch (innervating the septum in a left-to-right direction) that comes from the left bundle. It does not have a pattern seen with a biventricular pacemaker, as the initial waveform of the QRS complex in lead I is an R wave and not a Q wave. Hence the QRS complex has an intraventricular conduction delay. Despite the preceding ventricular pacing spike, this complex is not captured, *ie*, it is not in response to the pacing stimulus. The QT/QTc intervals are prolonged (420/480 msec) but are normal when the prolonged QRS complex duration is considered (400/455 msec).

There is a P wave (▲), without a pacemaker stimulus, before each of the QRS complexes associated with the short RR interval (●●). However, the P wave is negative in leads II, III, aVF, and V4–V6. They are not retrograde P waves as the preceding QRS complex (●) has a P wave before it and retrograde atrial activity does not occur when there is atrial activity associated with the preceding QRS complex. Retrograde atrial activity occurs with a junctional complex, ventricular paced complex (without initial atrial activity) or a ventricular complex. Hence these are premature atrial complexes. Each of the QRS complexes that follow the negative P wave are preceded by pacemaker stimulus (++). The AV delay (or PR interval) is 0.16 sec, identical to the AV delay of the pacemaker. Hence this is P-wave synchronous ventricular pacing (or atrial sensed, ventricular paced). The QRS complex duration is longer (0.16 sec) than the AV sequentially paced complex and it has a slightly different morphology, more typical of a LBBB with a broad R wave in leads I and V6 (→). This complex is due to complete ventricular captured by the pacing stimulus.

The QRS complex that is AV sequentially paced has a pseudofused morphology due to the fact that the native PR interval and AV delay of the pacemaker are about the same. Hence there is left ventricular activation that is primarily originating via the normal AV node–His-Purkinje system, with less activation via the RV pacemaker. The QRS complex of the premature atrial complex is completely captured and hence the complex is wider and more abnormal, with a typical LBBB morphology. This is due to the fact that left ventricular activation is via the pacemaker and not a result of AV node–His-Purkinje conduction. The reason for this is that the premature atrial impulses, arrives at the AV node before it has completely repolarized. As it is still partially refractory, conduction through the node is slower than normal. This is due to the property of the AV node called decrimental conduction, *ie*, in a nonsympathetic state the faster the impulses arrive at the AV node, the slower is conduction through this structure. As a result of the delay in impulse conduction through the AV node, the ventricular myocardium is completely activated by the pacemaker stimulus. ■

Notes

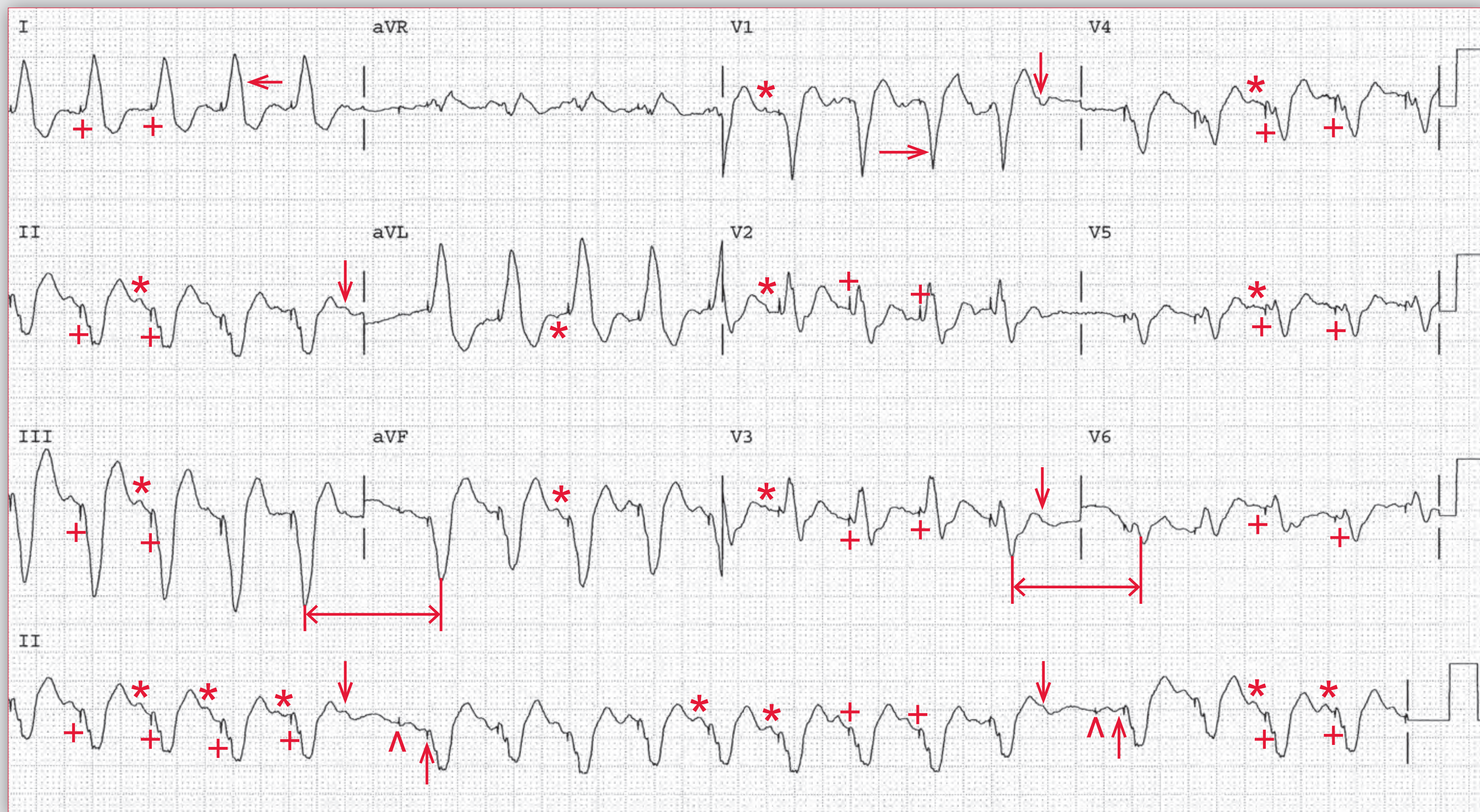
Core Case 23

A 74-year-old man who is known to have a dual-chamber pacemaker presents to the emergency department with a productive cough, fever of 103°F, and an elevated WBC count. A chest x-ray confirms the presence of a bilateral pneumonia. An ECG is obtained. The emergency department physician is concerned about the pacemaker and requests a cardiology consult.

What is the abnormality noted?

Is pacemaker function normal?

Is any additional therapy necessary?



ECG 23 Analysis: Sinus tachycardia, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous ventricular pacing), intermittent failure to sense the P wave (pseudo second-degree AV block)

There is a regular rhythm at a rate of 130 bpm, although there are two long RR intervals (↔) (pauses in the rhythm) noted. Each QRS complex is preceded by a P wave (*), which is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. Before each QRS complex there is a pacemaker stimulus (+). The QRS complex duration is increased (0.18 sec), and it has a left bundle branch block morphology with a broad R wave in leads I (←) and QS complex in lead V1 (→). Hence there is a dual-chamber pacemaker functioning in an atrial sensed, ventricular paced mode (or P-wave activated or synchronous ventricular pacing). The AV delay of the pacemaker is 0.14 sec. The QT/QTc intervals are prolonged (340/500 msec) but are normal when the prolonged QRS complex duration is considered (260/380 msec).

Before each pause there is an on-time sinus P wave (↓) seen that is not followed by a paced QRS complex. Therefore, this is a nonsensed P wave. The complex ending the pause has a pacemaker stimulus before the P wave (^) as well as before the QRS complex (↑). Hence this complex demonstrates AV sequential pacing.

The reason for the nonsensed P wave that does not result in a paced QRS complex (nonconducted) is that the underlying heart rate (sinus tachycardia) is at the upper rate limit of the pacemaker (*ie*, 130 bpm). Therefore, the pacemaker occasionally fails to sense the on-time P wave (↓) (which is occasionally at a rate slightly faster than 130 bpm). This is based upon the "blanking period" of the pacemaker determined by the PVARP (post-ventricular atrial refractory period), *ie*, the time after a ventricular complex that the pacemaker does not sense an atrial impulse. As a result of this blanking period, during which time the pacemaker does not sense a P wave, there is no ventricular stimulus and hence a pause. Importantly, if a P wave is sensed the pacemaker is committed to deliver a ventricular stimulus unless it is suppressed by spontaneous ventricular activity, *ie*, a premature ventricular complex or a native QRS complex resulting from AV conduction. This finding also means that the patient has complete AV or heart block, as if there were intact AV conduction, there would have been a native QRS complex following this nonsensed P wave, and the PR interval would be longer than the AV delay of the pacemaker. After the pause, there is an atrial pacemaker stimulus that occurs before the next regular sinus impulse would have occurred, and this is followed by a paced QRS complex—hence, AV sequential pacing. This pause is related to the lower rate limit of the pacemaker, which is determined by the interval between the last ventricular stimulus and the following atrial stimulus, *ie*, rate of 75 bpm. Therefore, the pacemaker function is normal.

Once the patient's pneumonia is treated and the sinus rate decreases, this abnormality will no longer be present, as at a slower sinus rate each P wave will be sensed, resulting in appropriate atrial sensing and ventricular pacing. ■

Notes

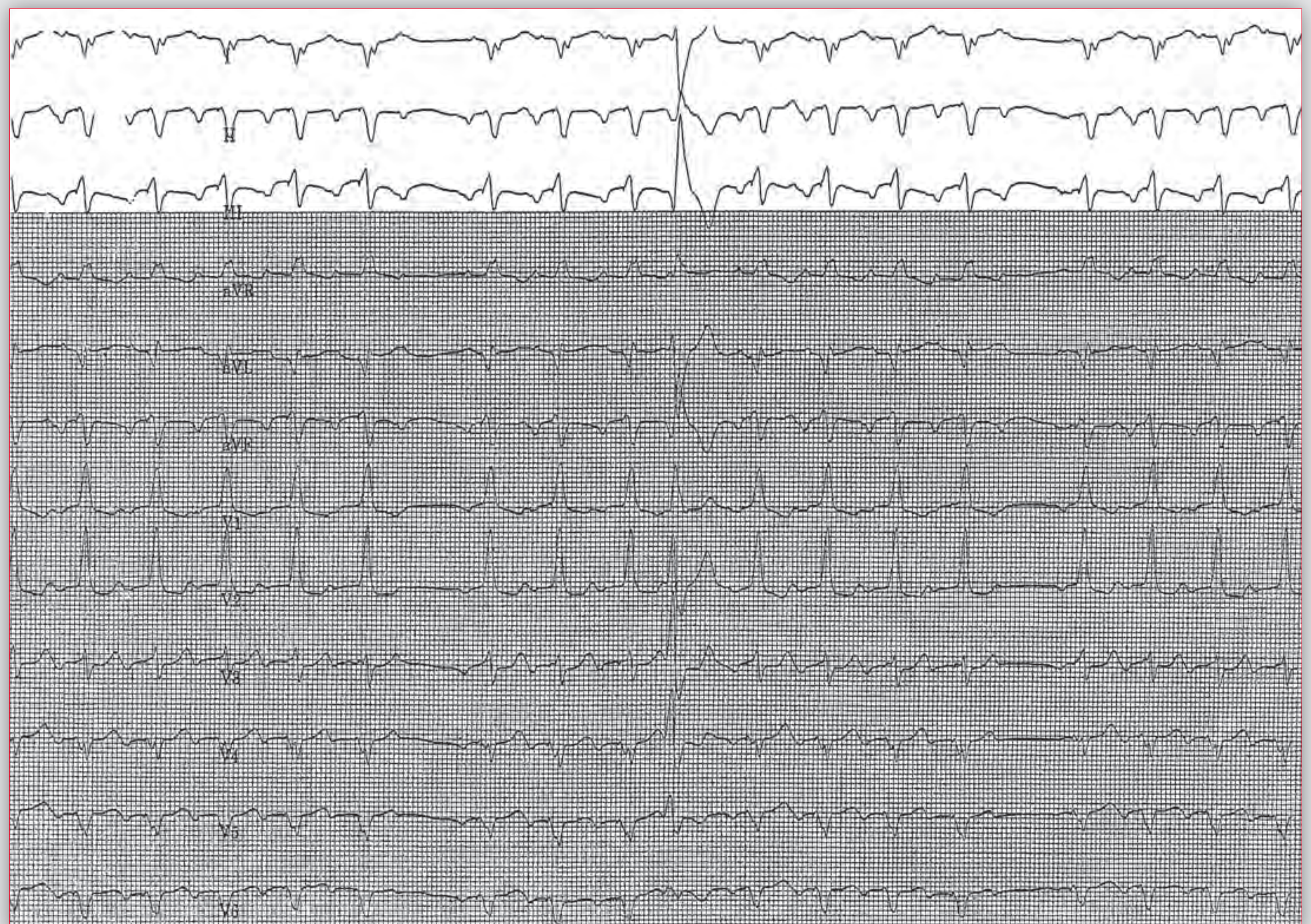
Core Case 24

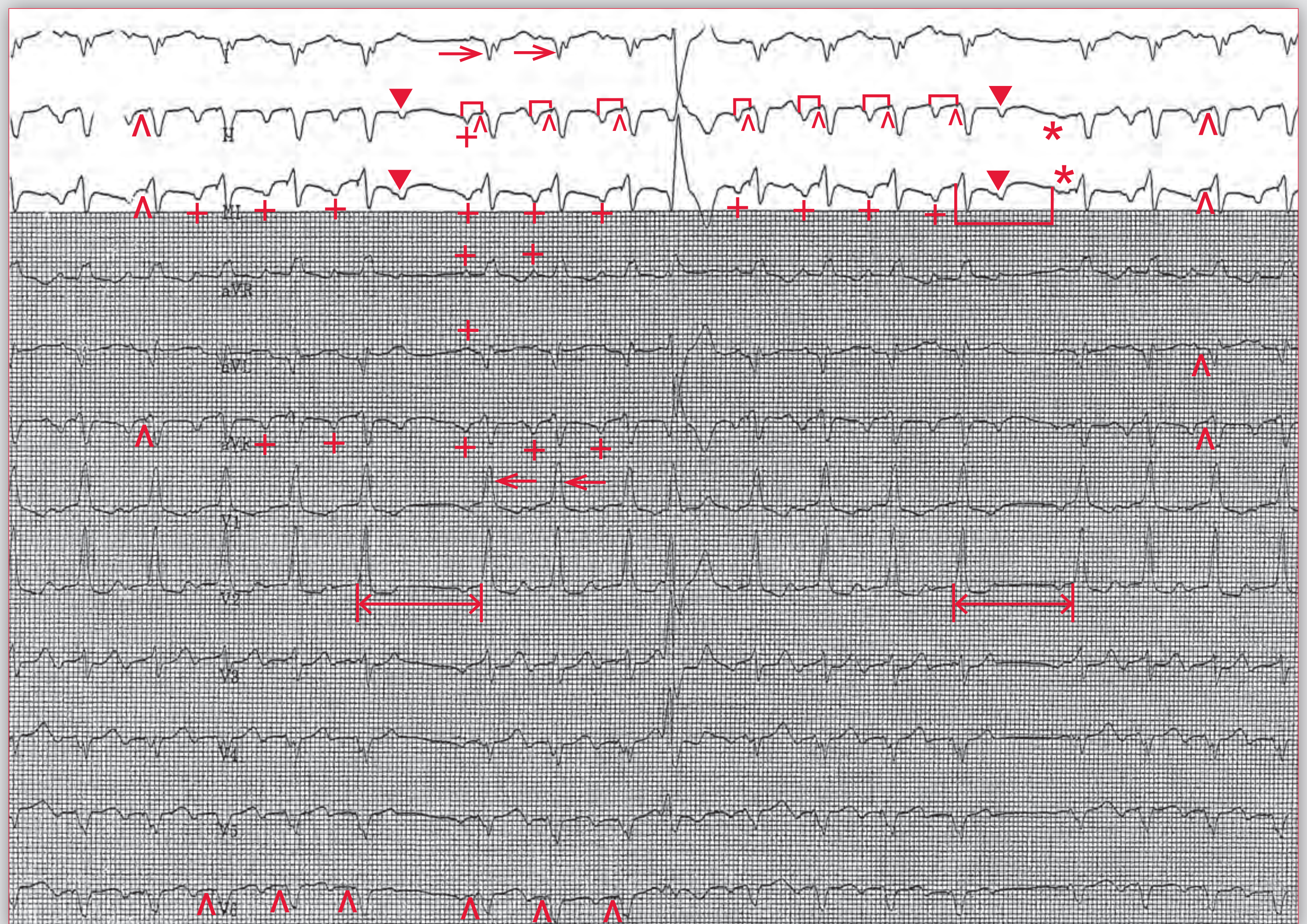
A 68-year-old man with a dual-chamber pacemaker is seen for a routine pacemaker check. It is noted that his heart rate is 120 bpm and hence an ECG is obtained.

What does this show?

What is the underlying rhythm?

Is pacemaker function normal?





ECG 24 Analysis: Atrial tachycardia, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous ventricular pacing), biventricular pacing, pseudo-Wenckebach (pacemaker-mediated Wenckebach), pseudo second-degree AV block

The rhythm is mostly regular with a rate of 120 bpm, although there are long RR intervals seen (↔). There are P waves (+) noted before each QRS complex. P waves are especially apparent during the long RR intervals (▼) as these P waves are nonconducted, *ie*, they are not followed by a QRS complex. The P waves are negative in leads II, aVF, and V4–V6. Hence this is not a sinus rhythm, but is an atrial tachycardia. There are pacing stimuli seen before each QRS complex (^). Therefore, this is a dual-chamber pacemaker functioning in an atrial sensed, ventricular paced (P-wave synchronous) mode. There is also an atrial stimulus (*) seen before the P wave following the second long RR interval, and the P wave has a different morphology compared the other P waves. Hence this is an AV sequentially paced complex. The QRS complex duration is increased (0.14 sec). However, the QRS complex morphology is not typical for right ventricular pacing, as there is a QS complex in lead I (→) and a tall R wave in lead V1 (←). This pattern is characteristic of a left ventricular or biventricular pacemaker. Lead I is the most important lead as it is the only R-L bipolar lead. Any impulse originating from the right generates an R wave in lead I while an impulse originating from the left generates a Q wave or QS complex in lead I. The QT/QTc intervals are prolonged (340/500 msec) but are normal when the prolonged QRS complex duration is considered (300/440 msec).

Although there is a pacemaker stimulus before each QRS complex, it can be seen that the PR interval (AV delay or interval between P wave and ventricular pacemaker stimulus) (⊓) of the pacemaker is not constant. Instead there is a progressive lengthening of the PR interval, *ie*, from 0.12 sec to 0.26 sec before the nonconducted P wave (▼). The presence of progressive PR interval lengthening and then a single nonconducted P wave is Mobitz type I or Wenckebach. When it is noted with a pacemaker, it is termed "pseudo-Wenckebach" or pacemaker-mediated Wenckebach.

The occurrence of this pattern is related to two programmable features of the pacemaker, *ie*, the post-ventricular atrial refractory period (PVARP) which determines the upper rate limit of the pacemaker (the fastest atrial rate sensed or tracked by the pacemaker resulting in a ventricular stimulus) and the AV delay of the pacemaker. These two parameters define the total atrial refractory period (TARP). If there is an underlying sinus or atrial rate that is close to the upper rate limit of the pacemaker (as is the case here, as most of the P waves are sensed and result in a ventricular stimulus), the atrial impulse will be sensed by the atrial channel (as the atrial impulse occurs after the PVARP). If the atrial impulse is sensed the pacemaker is committed to deliver a ventricular stimulus (unless it is inhibited by a spontaneous ventricular complex, *ie*, a premature ventricular complex or QRS complex resulting from AV conduction). However, if based on the AV delay (which may be short), delivery of a ventricular stimulus would violate the upper rate limit of the ventricular channel, the pacemaker waits until the upper rate limit is achieved before the ventricular stimulus is delivered. After the ventricular stimulus is delivered the PVARP starts again. If there is a stable atrial rate, the next atrial impulse is now closer to the PVARP and there is even a longer time before the ventricular stimulus can be

continues

delivered, accounting for a lengthening of the AV delay. This continues until the atrial impulse coincides with or is within the PVARP and is no longer sensed; hence there is no ventricular stimulus, *ie*, there is a nonconducted P wave. This is most frequently seen with biventricular pacing, since often the programmable AV delay is short, insuring that the pacemaker always captures the ventricles.

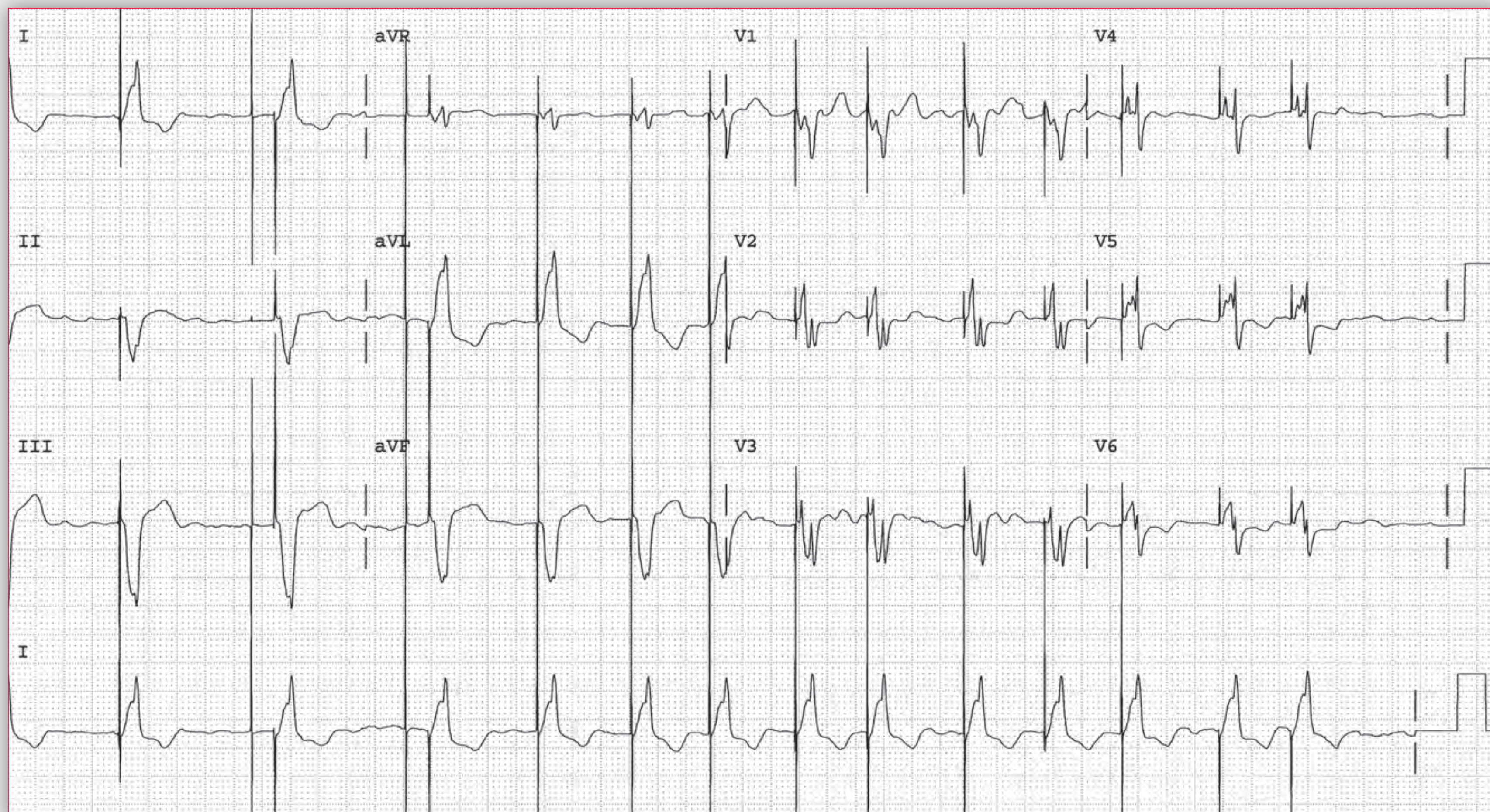
The two long RR intervals are the result of a nonsensed P wave (▼), as these two P waves are occurring with a coupling interval with the previous paced QRS complex that is slightly shorter, and as a result, they are now occurring within the PVARP and hence not sensed by the atrial channel. Therefore, there is no ventricular stimulus generated. After the second long RR interval, there is an AV sequentially paced complex, and the interval between the last ventricular stimulus and the atrial stimulus (⊔) is equal to the lower rate limit of the pacemaker (*ie*, 72 bpm). It can be seen that the atrial paced complex occurs before the next on-time atrial P wave would have occurred. After the first long RR interval, there is an on-time atrial P wave that occurs at a faster rate than the lower rate limit of the pacemaker and hence there is no atrial paced complex seen. ■

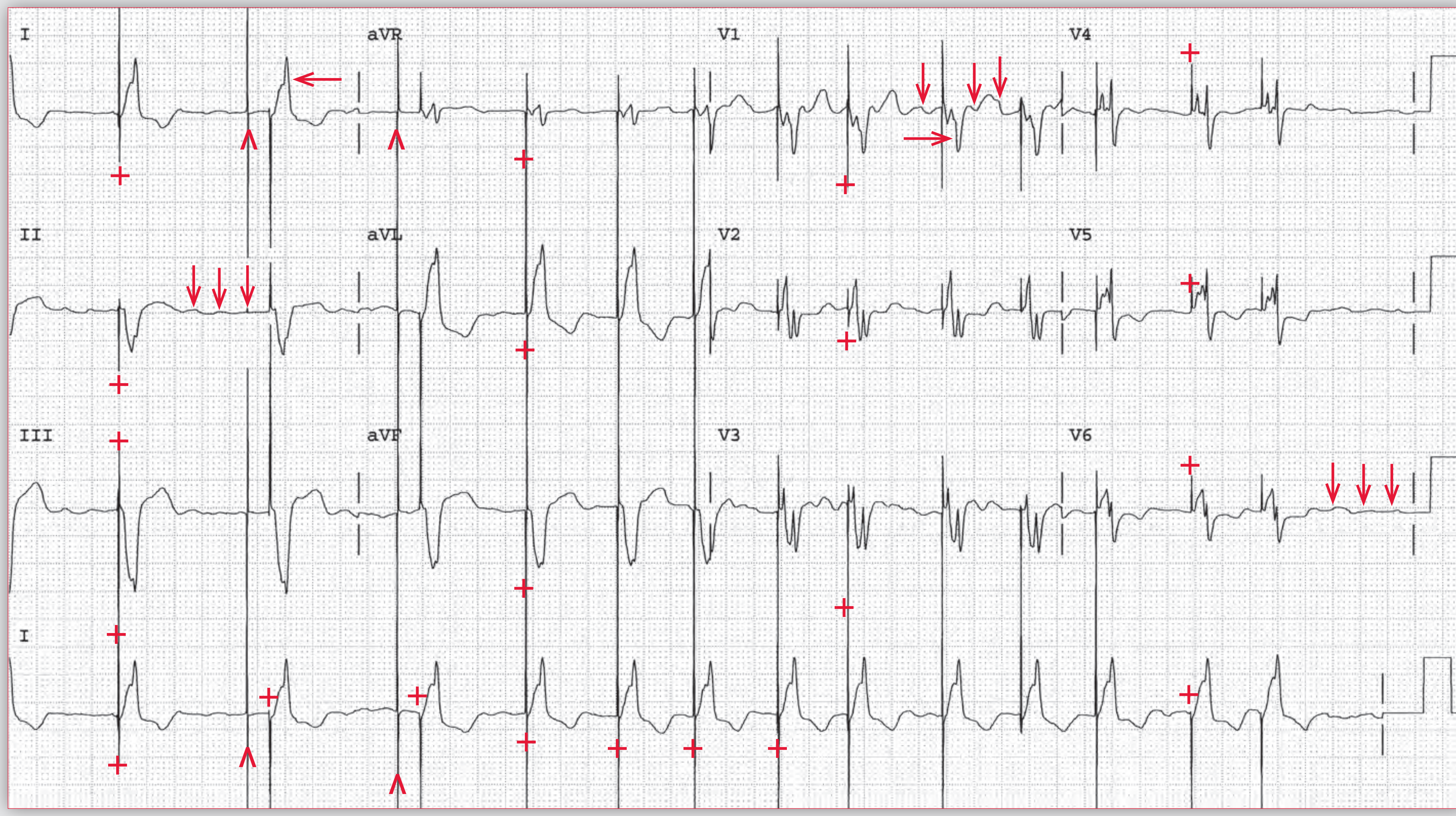
Core Case 25

A 62-year-old man, with a known sick sinus syndrome and pacemaker, presents to the emergency department with complaints of palpitations and an irregular heart rate. He states that his heartbeat has always been regular, although prior to the pacemaker it was very slow. An ECG is obtained.

What kind of pacemaker does he have?

Is the pacemaker function normal?





ECG 25 Analysis: Atrial fibrillation, P wave synchronous right ventricular pacing (atrial sensed-ventricular paced), intermittent AV sequential pacing

The rhythm is irregularly irregular. The average rate is 84 bpm. There are no organized P waves seen before or after the QRS complexes. However, there are rapid and irregular undulations of the baseline (↓), representing fibrillatory waves. Hence the underlying rhythm is atrial fibrillation. The QRS complex duration is prolonged (0.16 sec) with a left bundle branch block morphology with a broad R wave in lead I (←) and a QS complex in lead V1 (→). Each QRS complex is preceded by a pacemaker stimulus (+) (ventricular pacing). The second and third QRS complexes have a second pacemaker stimulus (^), which is an atrial stimulus. The interval between these two pacing stimuli, which is the AV delay of the pacemaker, is 0.16 sec. This is a dual-chamber pacemaker and is functioning mostly in an atrial sensed, ventricular paced mode. However, the second and third QRS complexes demonstrate AV sequential pacing as atrial activity was not sensed. The QT/QTc intervals are prolonged (400/470 msec) but are normal when the prolonged QRS complex is considered (340/400 msec).

As a result of atrial fibrillation that is associated with a rapid and irregular atrial rate, the atrial lead senses some but not all atrial activity. As the sensing is irregular, the ventricular output or ventricular response rate is also irregularly irregular. In addition, there are episodes where the atrial lead fails to sense any atrial activity, resulting in AV sequential pacing (*ie*, the second and third QRS complexes). However, the impulse from the atrial lead does not capture because of the rapid atrial rate, resulting in the atrial myocardium being continuously refractory. This is commonly observed with atrial fibrillation as this arrhythmia is due to multiple reentrant circuits in the right and left atria. Hence the atrial waveforms may only intermittently be sensed, depending upon where the atrial waveforms originate in relation to the location of the right atrial pacing lead. In addition, the amplitude of the atrial fibrillatory waves may be too low to be sensed, based on the sensitivity of the atrial channel. Not uncommonly all of the atrial fibrillatory waves are sensed and this will result in the pacemaker sensing all the atrial impulses. In this situation the ventricular pacing rate will be rapid and regular, at the upper rate limit of the pacemaker. An important programmable feature of the pacemaker is called "mode switching." Mode switching requires that the pacemaker is able to detect atrial events occurring within the PVARP even though it does not track these complexes. In this situation, if a rapid atrial rate is sensed by the atrial lead, the pacemaker automatically eliminates all atrial sensing (the atrial lead is deactivated) and the pacing mode changes to VVI, *ie*, a ventricular demand pacemaker. If there is no atrial sensing, the pacemaker is unable to track any atrial impulses, preventing the pacemaker from pacing at a rapid rate. If the pacemaker does not mode switch and the patient is tracking atrial fibrillation at a rapid rate, a magnet can be applied to the pacemaker. The magnet inactivates all sensing and hence the pacemaker functions in a DOO mode, *ie*, a fixed-rate pacemaker that is unable to sense any atrial or ventricular activity, but continuously delivers pacemaker stimuli at the lower rate limit of the pacemaker. ■

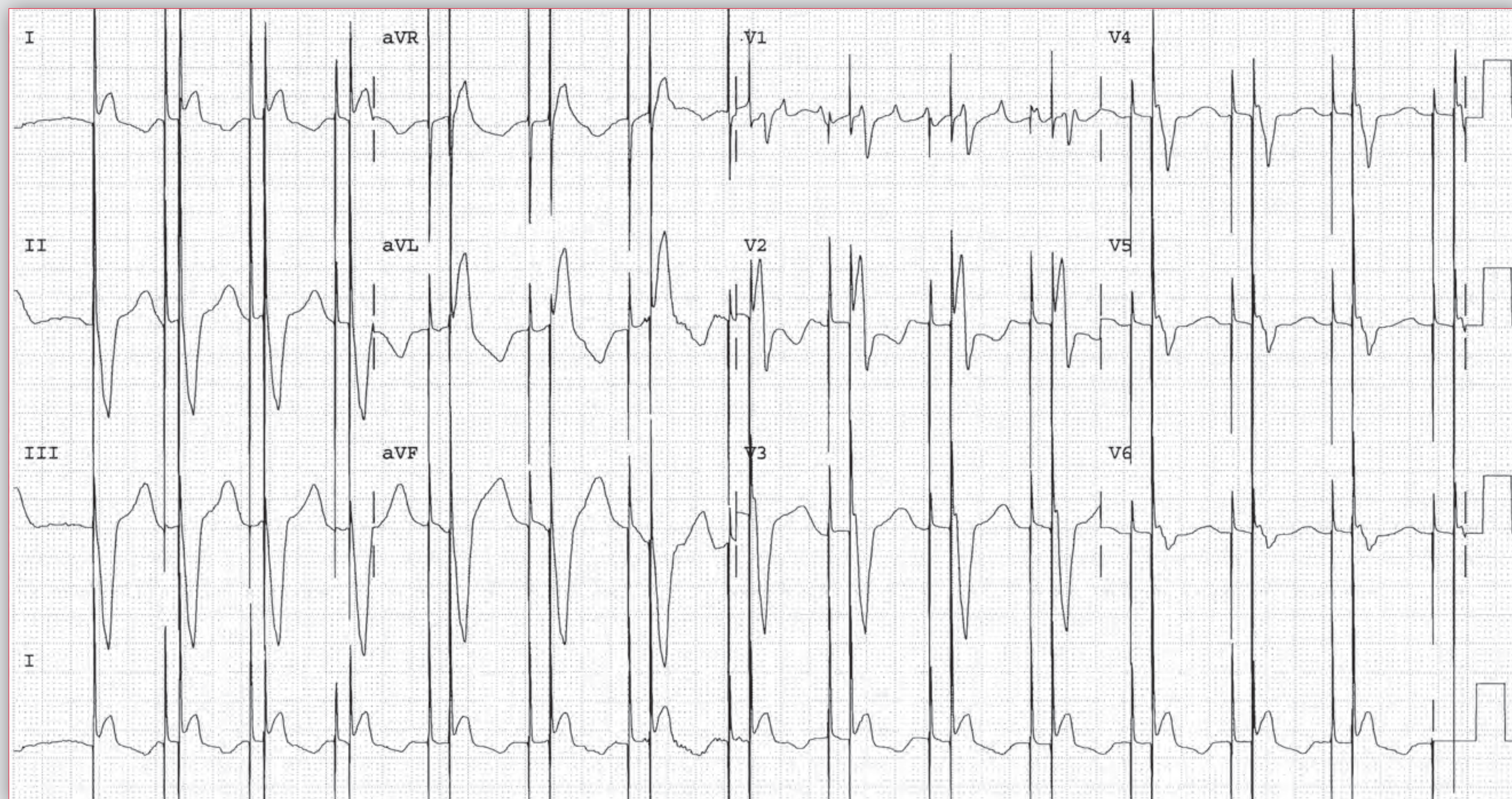
Notes

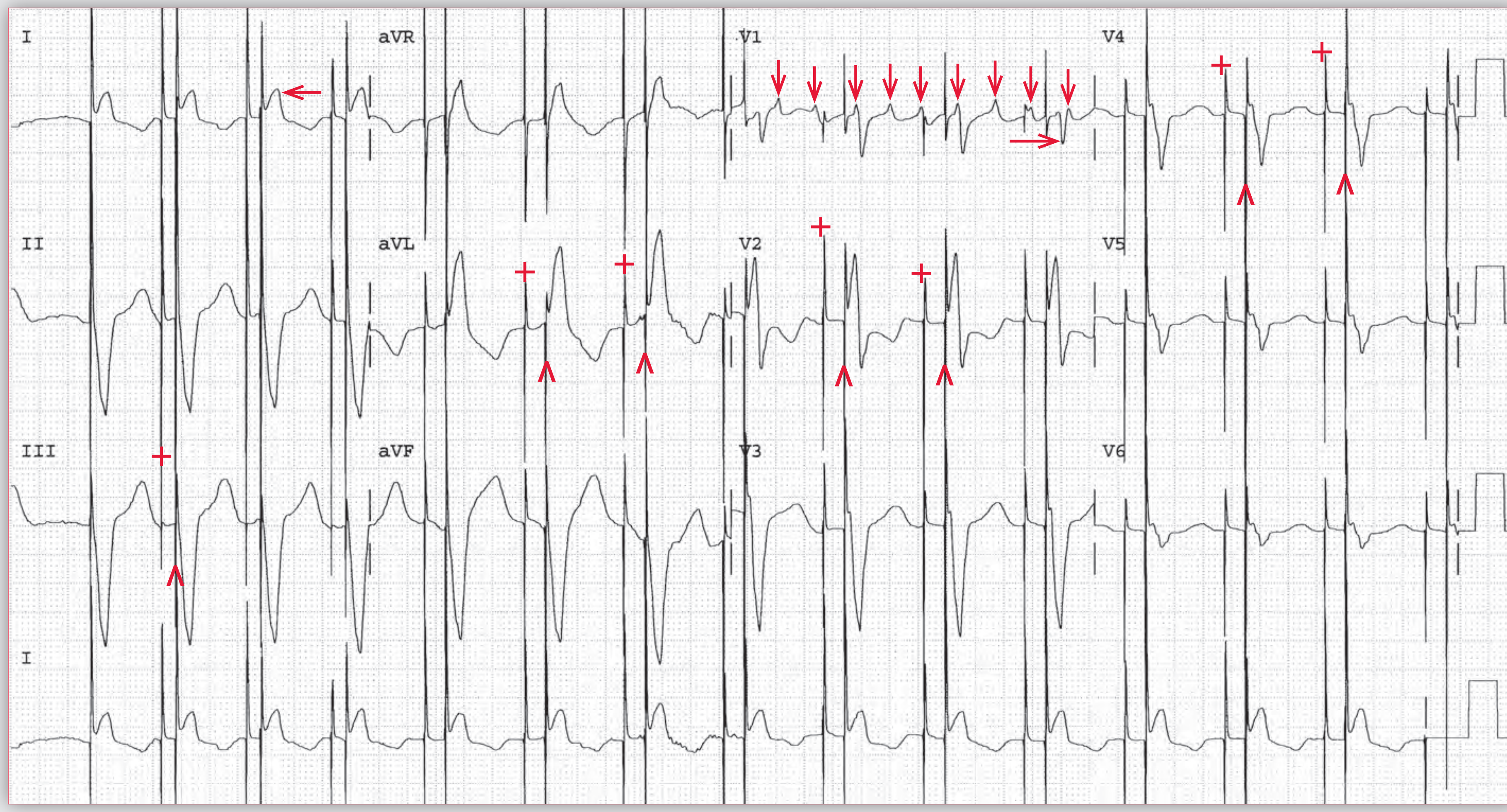
Core Case 26

A 76-year-old woman with a history of chronic obstructive pulmonary disease (COPD) and atrial fibrillation treated with sotalol presents to the hospital with a COPD exacerbation. She is begun on therapy with steroids, nebulizers, and multiple inhalers. An ECG obtained on the day of admission shows normal sinus rhythm with evidence of a pacemaker functioning in an atrial sensed, ventricular paced mode. On the following day telemetry reveals a change in her rhythm, and another ECG is obtained.

What is the change in her rhythm?

Is the pacemaker function normal?





ECG 26 Analysis: Atrial flutter, AV sequential pacing with failure of atrial lead to sense and capture

There is a regular rhythm at a rate of 86 bpm. There are two pacemaker stimuli before each QRS complex, *ie*, atrial (+) followed by ventricular (^). There is 100% ventricular capture. Hence this is AV sequential pacing with an AV delay of 0.14 sec. The QRS complex duration is increased (0.16 sec) and there is a left bundle branch block-like morphology with a broad R wave in lead I (←) and a QS complex in lead V1 (→), reflecting right ventricular pacing. The QT/QTc intervals are prolonged (440/530 msec), but are normal when the prolonged QRS complex duration is considered (380/455 msec). Noted, however, is that the underlying rhythm is atrial flutter, best seen in lead V1 where there are obvious flutter waves (↓) at a rate of 300. The atrial lead neither senses the atrial activity (as there are atrial pacing stimuli seen) nor captures the atrial myocardium (as the atrial flutter waves are present despite the atrial pacing stimulus). This is also best appreciated in lead V1.

The atrial lead fails to sense the atrial flutter, which is due to a reentrant circuit in the right atrium (RA). In this situation, the position of the atrial lead might preclude appropriate sensing of the atrial flutter waves. Another reason is that the RA lead is not able to sense the flutter waves because of their amplitude or other characteristics. This is based on the sensitivity of the atrial lead, *ie*, its sensitivity is low. In addition, the output from the atrial lead does not capture the atrium. This is due to the fact that the atrial flutter is too rapid to allow for a pacemaker stimulus to capture as the atrial myocardium is continuously refractory.

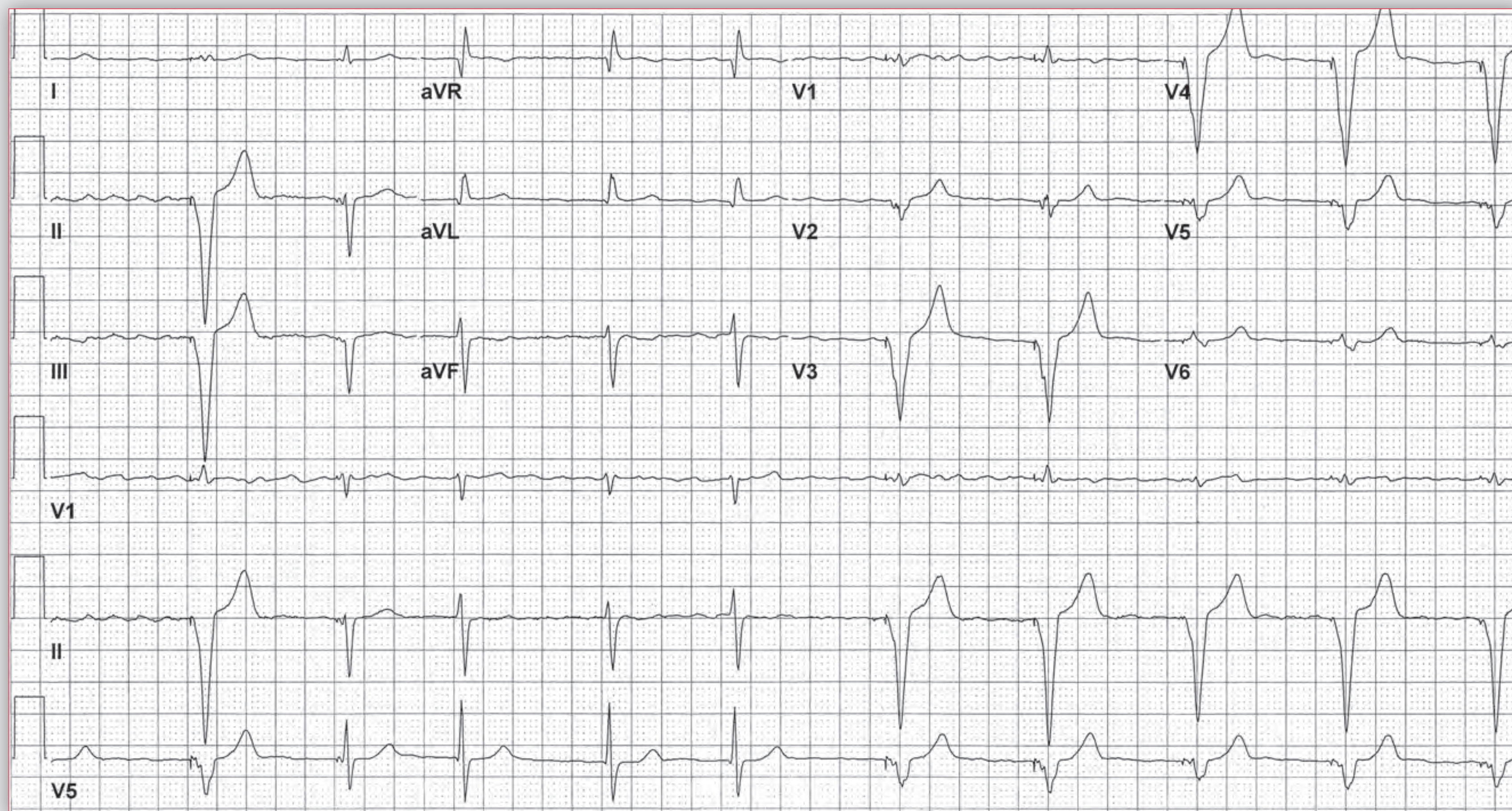
Sensing of the atrial flutter waves could result in ventricular pacing at a rapid rate, *ie*, at the upper rate limit of the pacemaker. However, there is a programmable feature of the pacemaker termed mode switching. In this situation, when a rapid atrial rhythm is sensed, the pacemaker automatically changes to a VVI pacing mode, *ie*, demand ventricular pacing. This avoids sensing of the rapid atrial waves by the RA lead and results in ventricular pacing at the upper rate limit of the pacemaker. ■

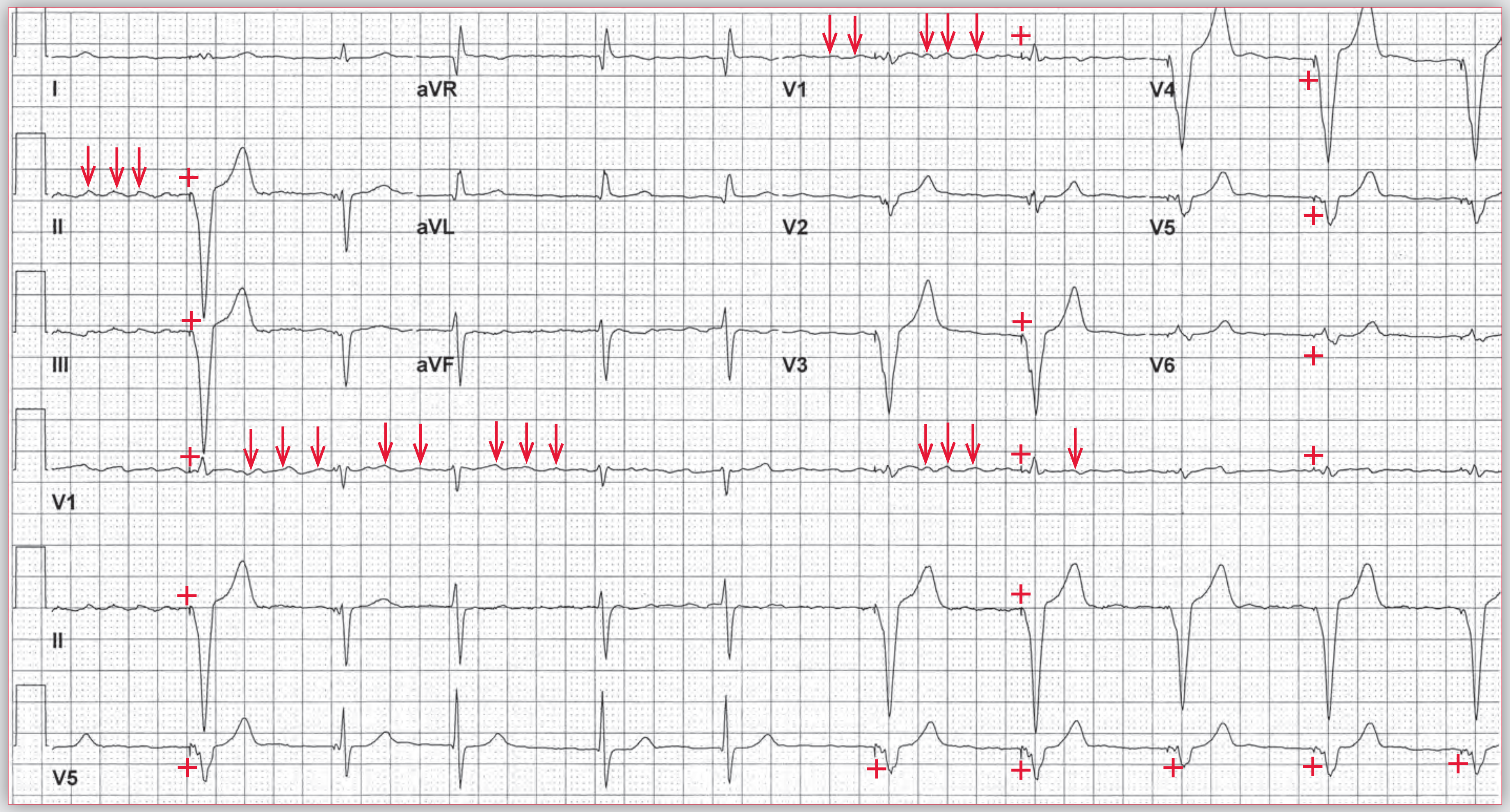
Notes

An 86-year-old man with a history of chronic atrial fibrillation and a pacemaker presents to an ophthalmology appointment prior to cataract surgery. Physical examination is unremarkable, but his blood pressure is low. Pulse is noted to be irregular, although periods of regularity are detected. An ECG is obtained, and the nurse practitioner evaluating the patient becomes concerned.

What is the underlying rhythm?

Is the pacemaker function normal?





ECG 27 Analysis: Atrial fibrillation, demand right ventricular pacing

There are wide QRS complexes (0.16 sec) that are at a regular rate of 60 bpm. These complexes have a pacemaker stimulus (+) before them. They are ventricular paced complexes with a left bundle branch morphology; hence, this a right ventricular pacemaker. There are also narrow QRS complexes (0.10 sec) that have irregularly irregular intervals. There are no obvious P waves seen before or after any of the QRS complexes, although there are irregular undulations (↓) of the baseline, particularly obvious in leads II, III, aVF, and V1. Hence the underlying rhythm is atrial fibrillation. The QT/QTc intervals are normal (380/380 msec). As a result of a rapid and irregular atrial rhythm (rates > 350–450 bpm), the ventricular response rate is irregular. In this case, whenever the ventricular response rate is > 60 bpm, the pacemaker is suppressed, and there is no ventricular output. When the ventricular rate is below 60 bpm, the pacemaker is activated, and there is fixed-rate ventricular pacing at a rate of 60 bpm. Hence this is a demand ventricular pacemaker (*ie*, VVI) in the presence of atrial fibrillation. It is also possible that this is a dual-chamber pacemaker that is programmed for mode switching, *ie*, if a rapid atrial rate is sensed, the pacemaker automatically switches to a VVI mode to avoid sensing a rapid atrial rate and pacing the ventricle at a rapid rate. Nevertheless, the pacemaker is functioning in a normal VVI mode. ■

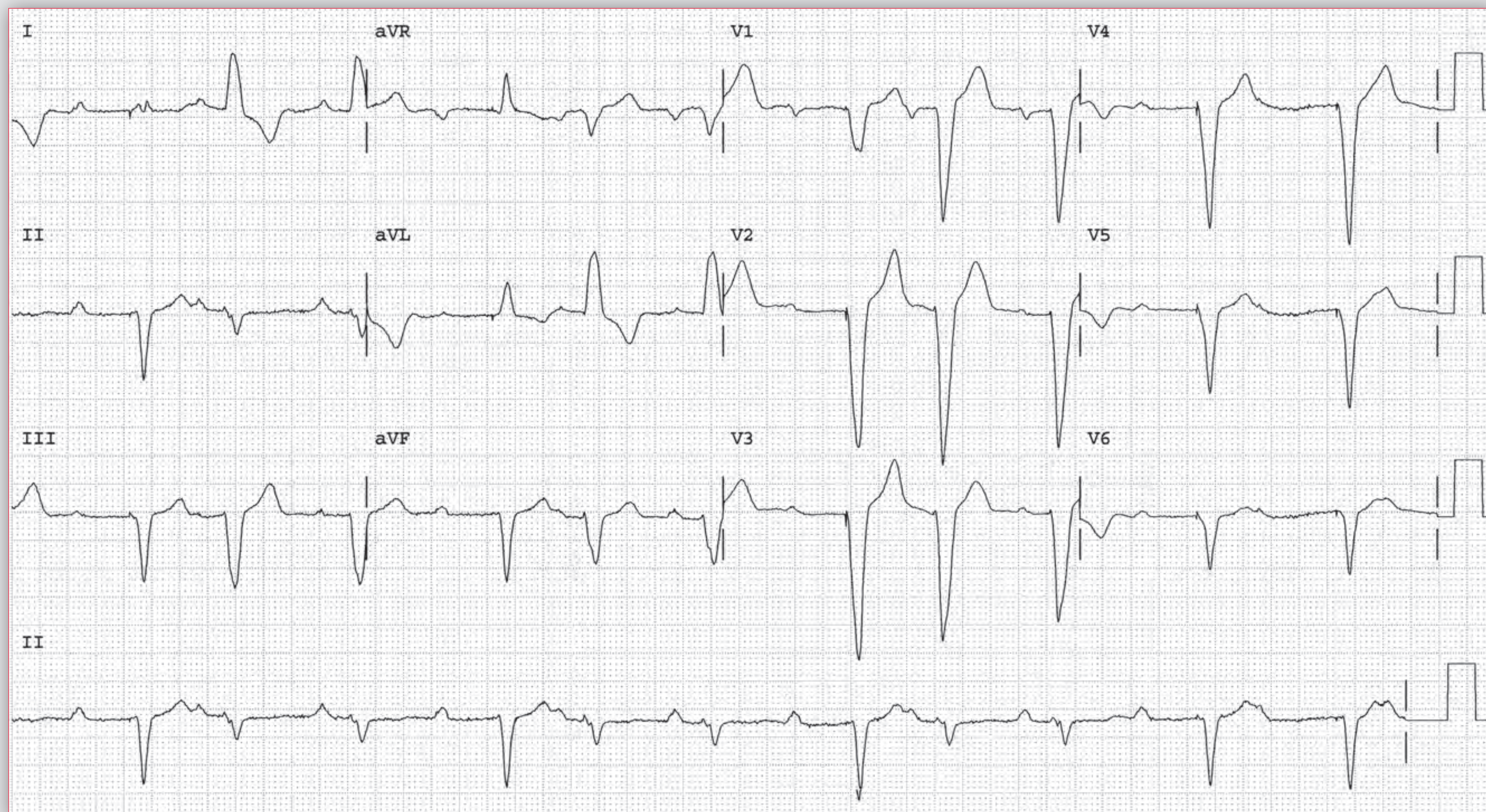
Notes

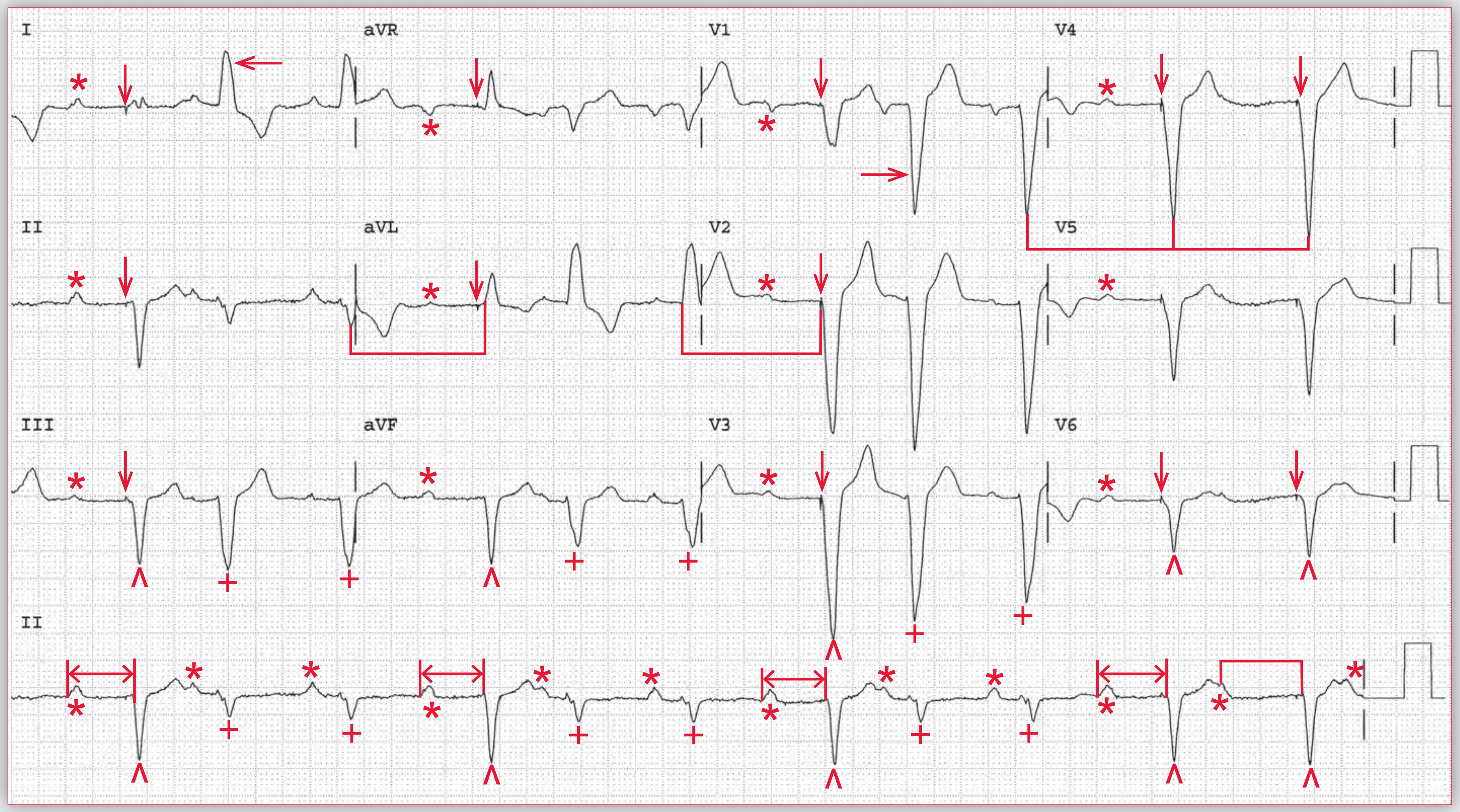
Core Case 28

A 71-year-old man presents to the emergency department because of episodes of lightheadedness and fatigue. He tells the attending physician that he had a history of these symptoms two years before, for which he received a pacemaker. An ECG is obtained and there is concern about pacemaker malfunction.

What is the underlying rhythm?

Is there evidence for pacemaker malfunction?





ECG 28 Analysis: Normal sinus rhythm, first-degree AV block, left bundle branch block, VVI pacing (demand right ventricular pacing)

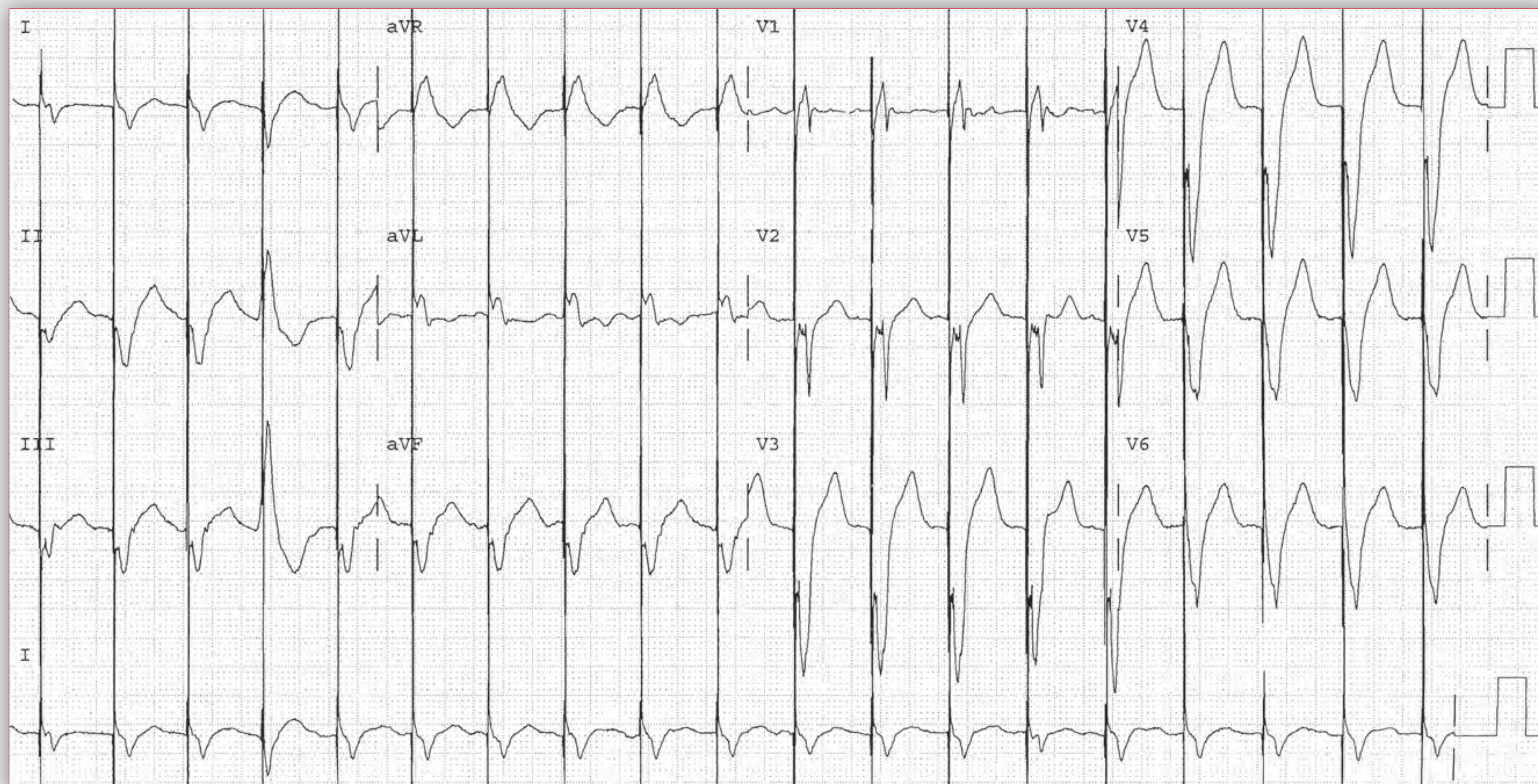
There is an underlying regular atrial rhythm with a rate of 68 bpm. There is a P wave (*) in front of each QRS complex. The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complexes have two different morphologies. The second, third, fifth, sixth, eighth and ninth complexes (+) have a QRS duration of 0.14 sec, and they have a typical left bundle branch block (LBBB) morphology with a broad R wave in lead I (←) and QS morphology in lead V1 (→). There is a stable PR interval (0.26 sec) associated with each of these QRS complexes. Hence these are sinus complexes with a first-degree AV block or conduction delay. The QT/QTc intervals are prolonged (460/490 msec) but are normal when the prolonged QRS complex duration is considered (420/450 msec). The first, fourth, seventh, tenth, and eleventh QRS complexes are slightly wider (duration 0.16 sec) (^) and they also have a QRS morphology is that typical for a LBBB. There is a pacing stimulus seen before each of these complexes (↓). Therefore, these are right ventricular paced complexes. There is also a P wave (+) before each of these QRS complexes with what appears to be a stable PR interval of 0.46 sec. This appears, therefore, to be P wave synchronous ventricular pacing with a long AV delay. However, the eleventh QRS complex that is paced follows the P wave with a much longer PR interval (0.60 sec) (⊓). Therefore, the pacemaker is not responding to the P wave before it, *ie*, it is not P-wave synchronous ventricular pacing. There is a fixed relation between the preceding sinus QRS complex (+) and the paced QRS complex (⊔), *ie*, a rate of 60 bpm. This is the same interval as with two sequential ventricular paced complexes (*ie*, complexes 10 and 11). Thus, the pacemaker is functioning in VVI mode or demand ventricular pacing. There is intermittent complete AV block, after which there is a ventricular paced beat (with a fixed-rate of 60 bpm) that occurs after a long and variable PR interval. Therefore, the pacemaker function is normal. ■

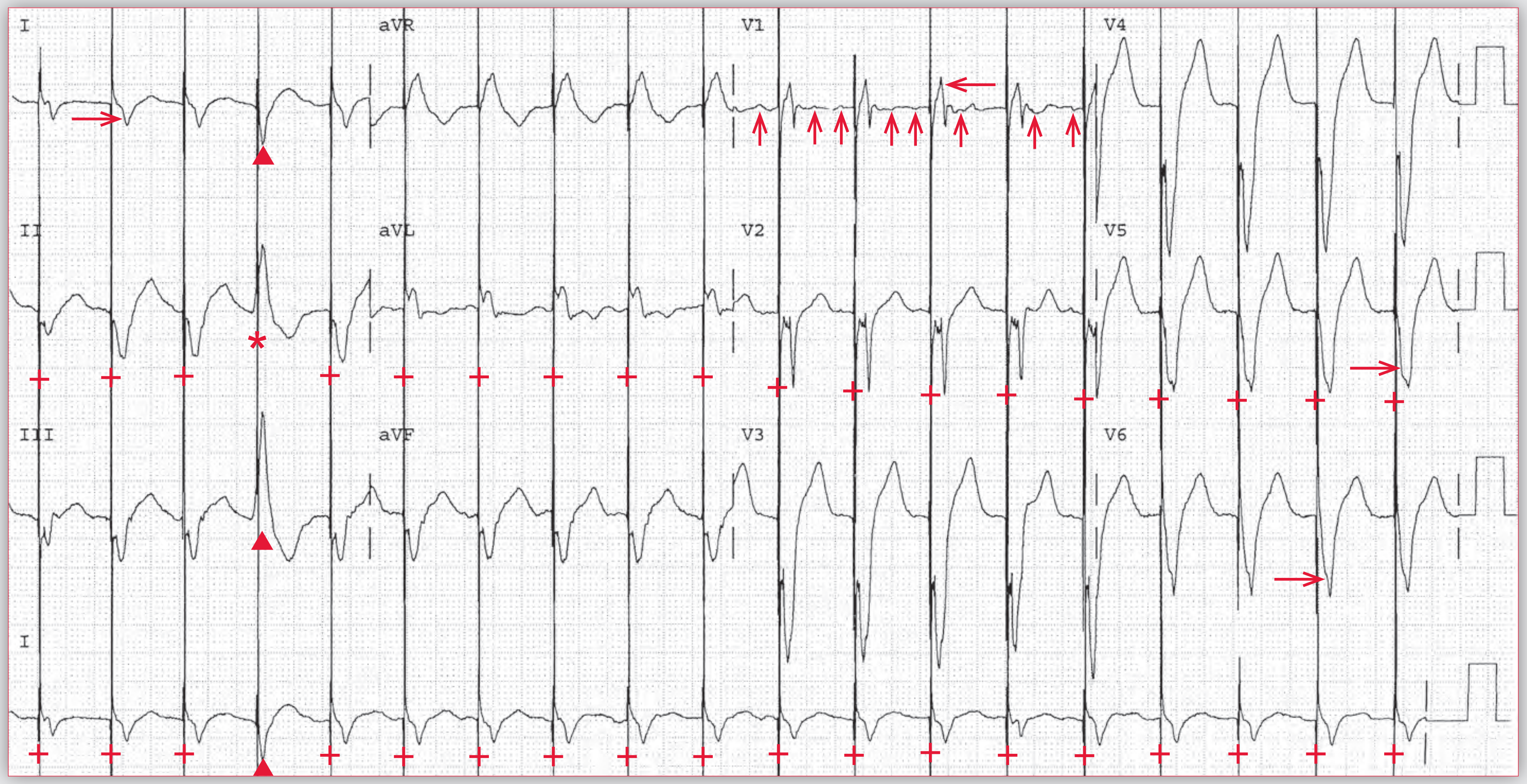
Notes

A 69-year-old woman presents to the emergency department complaining of palpitations for two days. She states that she has had paroxysmal atrial fibrillation in the past and as a result had a pacemaker inserted. The palpitations are different from what she has previously experienced, as her heart rate is now fast and regular while in the past her heart rate was fast but irregular. An ECG is obtained.

What is the underlying rhythm?

Is there a problem with the pacemaker?





ECG 29 Analysis: Atrial fibrillation, atrial activated ventricular pacing (at the upper rate limit of the pacemaker), biventricular pacing, premature ventricular complex

There is a regular rhythm at a rate of 125 bpm. The QRS complexes duration is prolonged (0.16 sec) and there is a pacing stimulus (+) before each QRS complex. Hence this is a ventricular paced rhythm. The QT/QTc intervals are prolonged (380/550 msec), but are only slightly prolonged when the prolonged QRS complex duration is considered (320/460 msec). There are no obvious P waves before or after any QRS complex; however, irregular and rapid undulations (↑) of the baseline can be seen, particularly in lead V1. Therefore, the underlying rhythm is atrial fibrillation. Since the ventricular pacing rate is 125 bpm, there must be atrial sensing resulting in ventricular pacing. Therefore, there is a dual-chamber pacemaker present with a right atrial lead that is sensing the atrial impulses and a ventricular lead that results in ventricular pacing. As the underlying rhythm is atrial fibrillation that has an atrial rate > 350–450 bpm, the atrial lead is sensing these rapid atrial impulses and pacing the ventricle at the upper rate limit of the pacemaker. Hence the ventricular rate is rapid and regular. Therefore, the dual-chamber pacemaker is functioning normally in an atrial sensed, ventricular paced mode. The fourth QRS complex (▲) has a different morphology and is slightly earlier. It is a premature ventricular complex that is not sensed by the ventricular lead; hence, there is an on-time ventricular pacemaker stimulus seen after the onset of the premature complex (*). Premature ventricular complexes are often not sensed by a lead as the premature complex may originate from a part of the myocardium that is a long distance from the ventricular lead.

In addition, the QRS morphology of the paced beats is not typical for a right ventricular (RV) pacemaker, *ie*, it does not have a left bundle branch block (LBBB)-like morphology, *ie*, a broad R wave in lead and a QS complex in lead I. In contrast, there is a broad R wave in V1 (←) and QS complex in leads I and V5–V6 (→). This is more like a right bundle branch block and indicates that activation is going from left to right. The most important lead is lead I, which is a bipolar lead, that sees the impulse as it travels from the right to left arm. With a RV pacemaker there should always be a broad R wave in this lead (the impulse travels from right to left). Lead I has a QS complex, which indicates that the ventricular impulse is traveling from left to right; the impulse is initiated in the left ventricle (LV). This pattern is typical for an impulse originating from the LV, *ie*, a biventricular pacemaker, which is often implanted into patients with drug refractory heart failure and a widened QRS complex duration, primarily as a result of a left bundle branch block (LBBB), to restore synchronous contraction of the LV, *ie*, cardiac resynchronization therapy (CRT). In addition to the right atrial and RV pacing lead, a pacing lead is also inserted into the coronary sinus over the LV. With a LBBB, there is dyssynchronous ventricular contraction, *ie*, the septum contracts before the posterior wall of the LV. With CRT, the initial activation is of the posterior wall of the LV that now contracts simultaneously with septal contraction, hence resulting in restoration of synchronous ventricle contraction.

The primary indication for the use of CRT (with or without an implantable cardioverter-defibrillator [ICD]) includes patients who have a LV ejection fraction (LVEF) ≤ 35%, a QRS duration greater than or equal to 0.15 sec due to a LBBB, sinus rhythm, New York Heart Association

continues

(NYHA) functional class II or III or ambulatory class IV heart failure symptoms despite optimal recommended medical therapy. CRT (with or without an ICD) may be useful, and is a reasonable consideration for patients who have LVEF ≤ 35%, NYHA functional class II or III or ambulatory class IV heart failure symptoms on optimal recommended medical therapy with a QRS duration greater than or equal to 0.15 sec and a non-LBBB pattern or those with a QRS complex duration between 0.12 to 0.15 sec, atrial fibrillation that requires ventricular pacing or atrial fibrillation with reasonable rate control (either from AV nodal ablation or pharmacologic therapy.

The pacemaker is functioning normally as it is appropriately tracking atrial impulses and pacing the ventricles. However, as the rhythm is atrial fibrillation, it is not sensing all of the atrial impulses but tracking them and responding to the atrial impulses with a ventricular output only at the upper rate limit of the pacemaker, accounting for the presence of a regular paced rhythm. As the pacemaker serves as a second pathway between the atrial and ventricles, therapies that block the AV node, which usually work to slow the ventricular rate of atrial fibrillation, will not be effective. To avoid this situation, there is a programmable feature of the pacemaker that is termed "mode switching." When the atrial lead senses rapid atrial activity, there is an automatic switching of the pacing mode to VVI. The atrial lead is inactivated and no longer has sensing capabilities. There is still ventricular sensing, and the pacemaker functions as a ventricular demand pacemaker. In this situation, AV nodal blocking agents to slow the ventricular rate will be effective as there is now normal impulse conduction via the AV node.

In the absence of mode switching, the rapid ventricular pacing rate can be treated by placing a magnet over the pacemaker. When this is done, all sensing is inactivated and the pacemaker functions in a DOO mode, *ie*, it is a non demand or fixed-rate pacemaker. It will deliver atrial and ventricular stimuli at the lower rate limit of the pacemaker. As the atria are being activated at rapid rates, the atrial impulse will not capture the atrial myocardium. However, there may be intermittent ventricular capture if the myocardium is able to respond, based on its refractory period and the timing of spontaneous QRS complexes resulting from impulses conducted via the AV node. ■

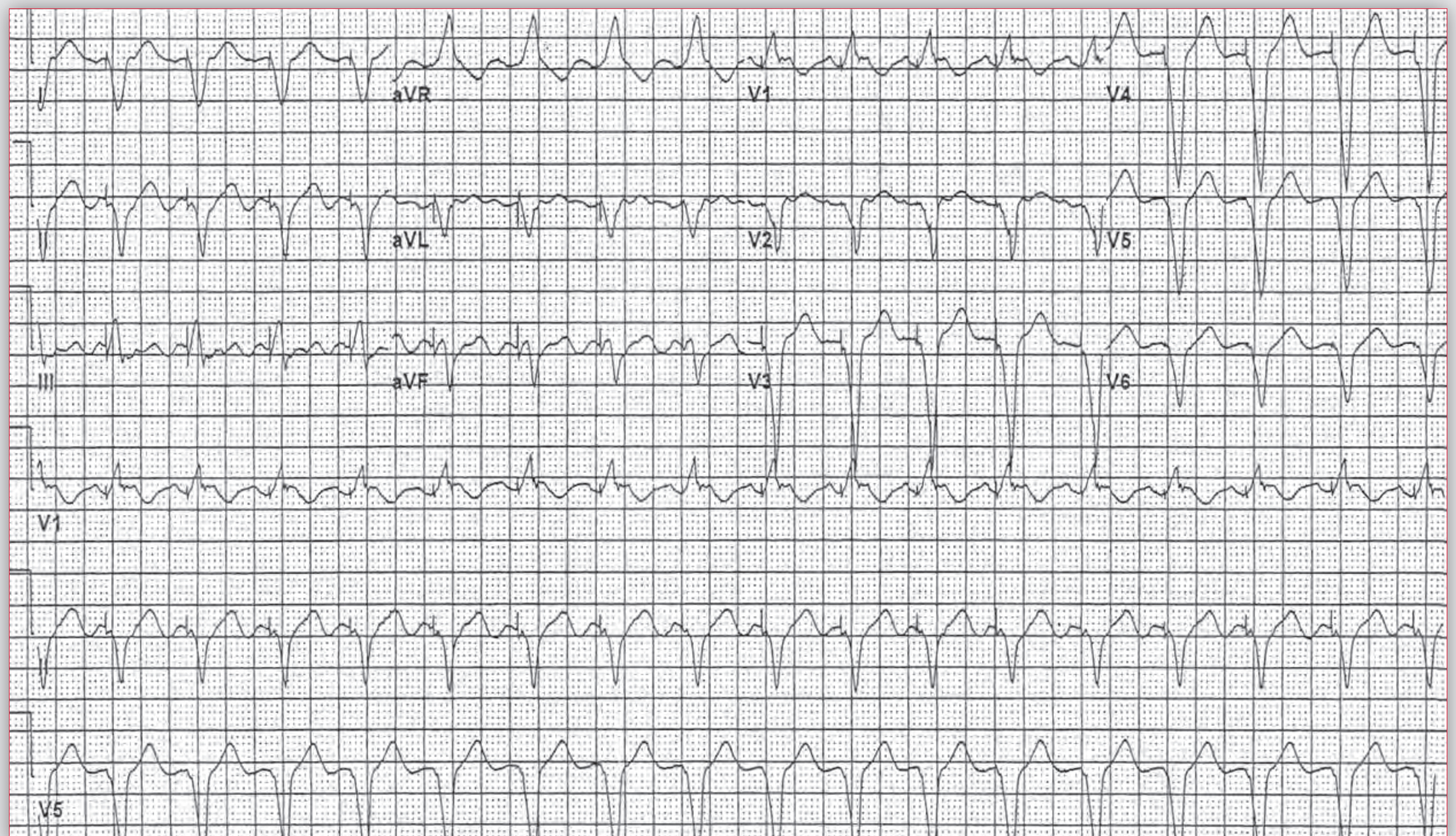
Core Case 30

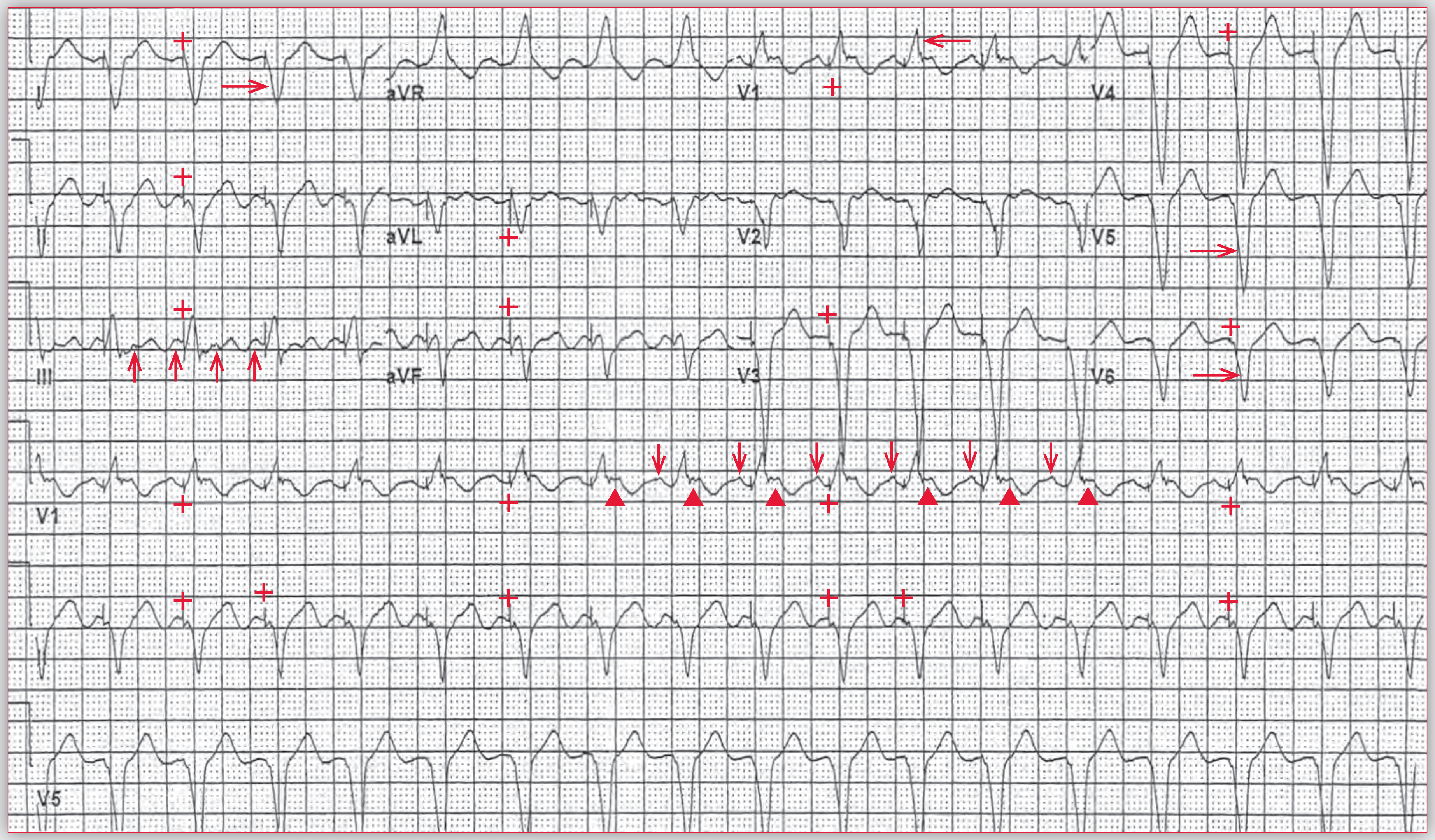
A 74 year-old-man with a history of paroxysmal atrial fibrillation on amiodarone therapy presents to his primary care provider with complaints of sensing his heart beating rapidly, similar to what he experienced with the atrial fibrillation. He does have an implantable cardioverter-defibrillator (ICD) that was placed two years before because of a low left ventricular ejection fraction (LVEF). He is uncertain about what type of ICD was placed. When seen, his physical examination was normal except for a regular pulse at a rate of 112 bpm. An ECG is obtained.

What type of pacemaker does this patient have?

It is functioning normally?

What is the underlying rhythm?





ECG 30 Analysis: atrial flutter, biventricular pacemaker, atrial sensed, ventricular paced (P-wave synchronous ventricular pacing) tracking atrial flutter with 2:1 block

There is a regular rhythm at a rate of 112 bpm. The QRS complex duration is increased (0.14 sec). The QT/QTc intervals are prolonged (380/520 msec) but are only slightly prolonged when the prolonged QRS complex duration is considered (340/460 msec). There is a pacemaker stimulus (+) before each QRS complex. Hence this is ventricular paced rhythm. There is atrial activity noted (↓) before each QRS complex, particularly obvious in lead V1. However, a second atrial wave (▲) can be seen immediately after the QRS complex. The PP interval is regular at a rate of approximately 220 bpm. The underlying rhythm appears to be an atrial tachyarrhythmia and every other P wave is sensed by the atrial lead resulting in a ventricular stimulus and QRS complex. Therefore, there is 2:1 AV conduction, *ie*, the pacemaker is tracking every other P wave. Hence this is a dual-chamber pacemaker functioning as atrial sensing and ventricular pacing (P-wave synchronous or P-wave activated ventricular pacing). The etiology of the atrial tachyarrhythmia is not certain, *ie*, atrial tachycardia or atrial flutter with 2:1 AV block. However, the atrial waveforms in leads II, III, and aVF appear to have a continuously undulating baseline (↑), making atrial flutter more likely. The atrial rate is slower than is usually seen with atrial flutter (*ie*, 260–320 bpm), and this may be the result of the amiodarone, which prolongs atrial refractoriness and does cause a slowing of the atrial flutter rate.

In addition, the QRS morphology of the paced beats is not typical for a right ventricular (RV) pacemaker, *ie*, it does not have a left bundle branch block (LBBB)-like morphology, *ie*, a broad R wave in lead I and a QS complex in lead I. In contrast, there is a broad R wave in V1 (←) and QS complex in leads I and V5–V6 (→). This is more like a right bundle branch block and indicates that activation is going from left to right. The most important lead is lead I; there should always be a broad R wave if this were a RV pacemaker, as it is the only bipolar lead that looks at impulses going left to right or right to left. As there is a negative QRS complex in this lead, ventricular activation is going from left to right, *ie*, initiated in the left ventricle (LV). Therefore, this pattern is typical for an impulse originating from the LV, *ie*, a biventricular pacemaker, which is often implanted into patients with drug refractory heart failure and a widened QRS complex duration, primarily as a result of a left bundle branch block, to restore synchronous contraction of the LV, *ie*, cardiac resynchronization therapy (CRT). In addition to the right atrial and RV pacing lead, a pacing lead is also inserted into the coronary sinus over the LV. With a LBBB, there is dyssynchronous ventricular contraction, *ie*, the septum contracts before the posterior wall of the LV. With CRT, the initial activation is of the posterior wall of the LV that now contracts simultaneously with septal contraction, hence resulting in restoration of synchronous ventricle contraction.

The primary indication for the use of CRT (with or without an ICD) includes patients who have a LVEF ≤ 35%, a QRS duration greater than or equal to 0.15 sec due to a LBBB, sinus rhythm, New York Heart Association (NYHA) functional class II or III or ambulatory class IV heart failure symptoms despite optimal recommended medical therapy.

continues

CRT (with or without an ICD) may be useful and is a reasonable consideration for patients who have LVEF ≤ 35%, NYHA functional class II or III or ambulatory class IV heart failure symptoms on optimal recommended medical therapy with a QRS duration greater than or equal to 0.15 sec and a non-LBBB pattern or those with a QRS complex duration between 0.12 to 0.15 sec, atrial fibrillation that requires ventricular pacing or atrial fibrillation with reasonable rate control (either from AV nodal ablation or pharmacologic therapy).

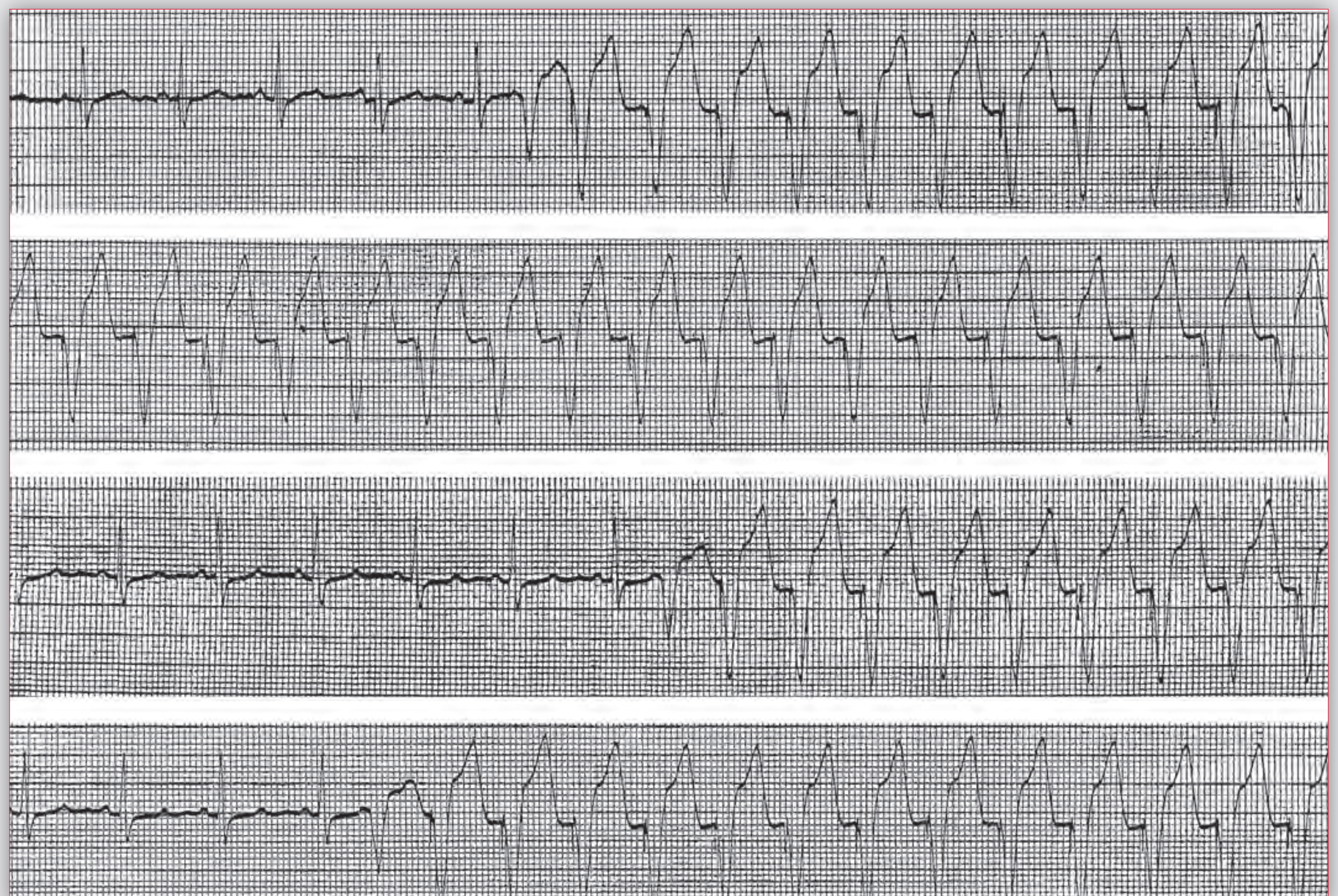
The pacemaker is functioning normally and is tracking the atrial flutter appropriately, resulting in a ventricular stimulus. As the atrial rate is far faster than the upper rate limit of the pacemaker, only every other atrial waveform is sensed, resulting in a ventricular stimulus, *ie*, a rate of 112 bpm. This is likely below the upper rate limit of the pacemaker. As the pacemaker represents another AV connection that cannot be influenced by AV nodal blocking drugs, the heart rate will be fixed. However, the use of a magnet, which inactivates atrial sensing, will change the pacemaker to a DOO mode, and hence there will be fixed-rate ventricular pacing at the lower rate limit of the pacemaker. This will eliminate atrial tracking, and hence the ventricular response rate will be solely related to the ability of the AV node to conduct. Hence AV nodal blocking agents will be of benefit to control the ventricular rate. However, as the pacemaker is now fixed rate, there will be atrial and ventricular stimuli occurring at the lower rate limit of the patient, not sensing the spontaneous P wave or QRS complex (as there is no longer any sensing). In addition it will only capture when the myocardium has recovered and is capable of responding to an electrical stimulus; this will appear as failure of the pacemaker to capture.

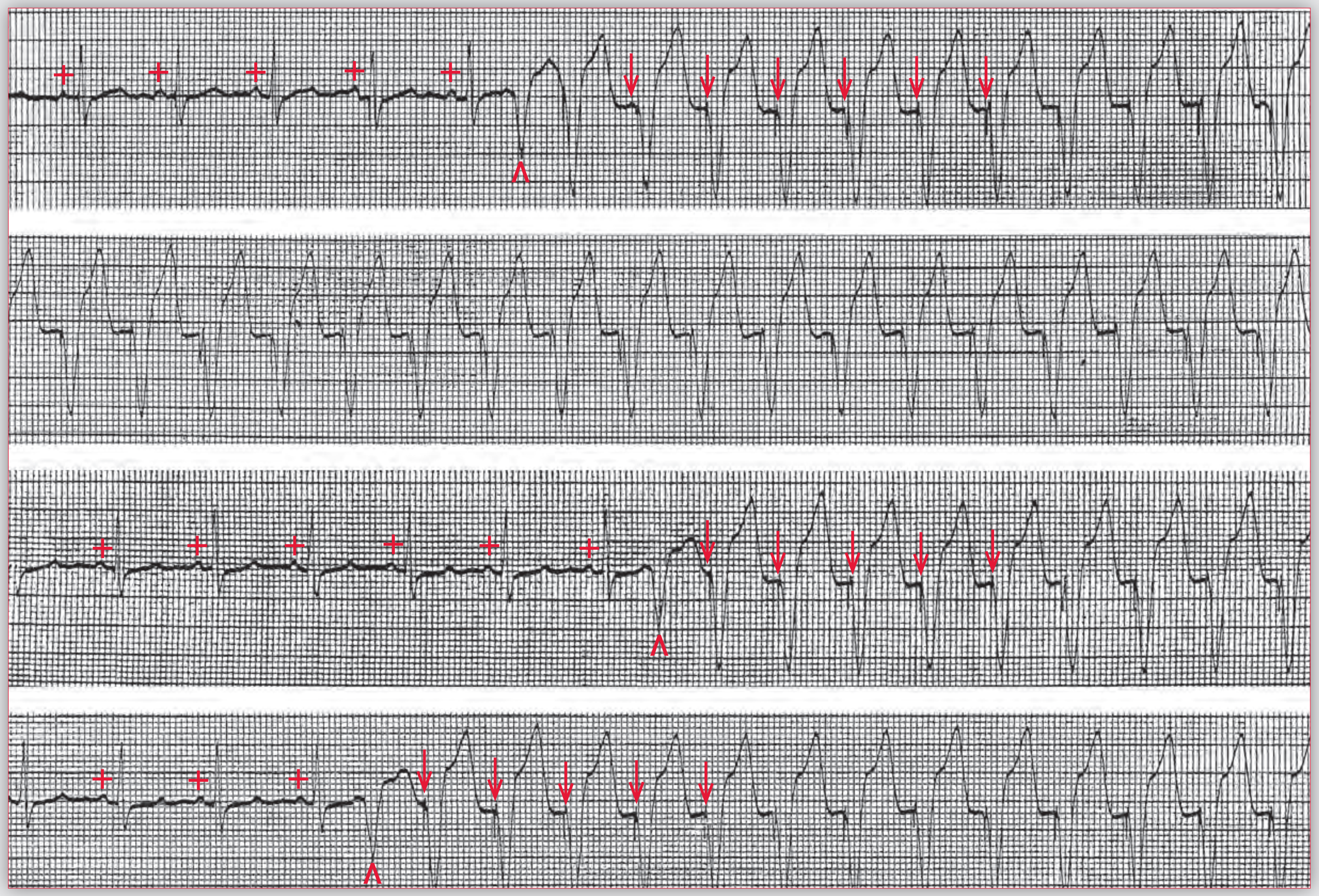
Alternatively, pacemakers may have a programmable feature known as "mode switching" in which sensed rapid atrial activity will result in the pacemaker switching to a VVI mode, or a demand ventricular pacemaker. In this mode, there is no atrial sensing or atrial output, but only a ventricular output whenever the intrinsic heart rate falls below the lower rate limit of the pacemaker. In this situation, the ventricular response rate is entirely dependent upon impulse conduction through the AV node. ■

Core Case 31

A 78-year-old man with a history of complete heart block and the insertion of a dual-chamber pacemaker presents to the emergency department with palpitations. He is placed on telemetry, and ECG strips are obtained during an episode of palpitations.

What do the ECGs show, and what is the mechanism for the tachycardia?





ECG 31 Analysis: Normal sinus rhythm, premature ventricular complex with VA conduction, pacemaker-mediated tachycardia

There are three separate rhythm strips. The first and second strips are continuous, and the third and fourth strips are individual. The first strip shows six QRS complexes that have a normal duration (0.08 sec) and are preceded by P waves (+). These are sinus complexes. The seventh QRS complex (^) is early, without a preceding P wave, and it is wide (0.14 sec). This is a premature ventricular complex (PVC). Following the PVC, there is a regular wide complex rhythm at a rate of 130 bpm. There is a ventricular pacing stimulus (↓) seen before each QRS complex. This is a typical pattern for a pacemaker-mediated tachycardia, which is induced by the PVC. There is a similar pattern seen on the third strip. There are 7 sinus complexes (+) followed by a PVC (^) that initiates a wide complex tachycardia at a rate of 130 bpm; there is a pacing stimulus (↓) seen before each QRS complex. On the fourth strip, there are three sinus complexes (+), followed by a PVC (^) that initiates a wide complex rhythm at a rate of 130 bpm; pacemaker spikes (↓) are seen before each of the QRS complexes.

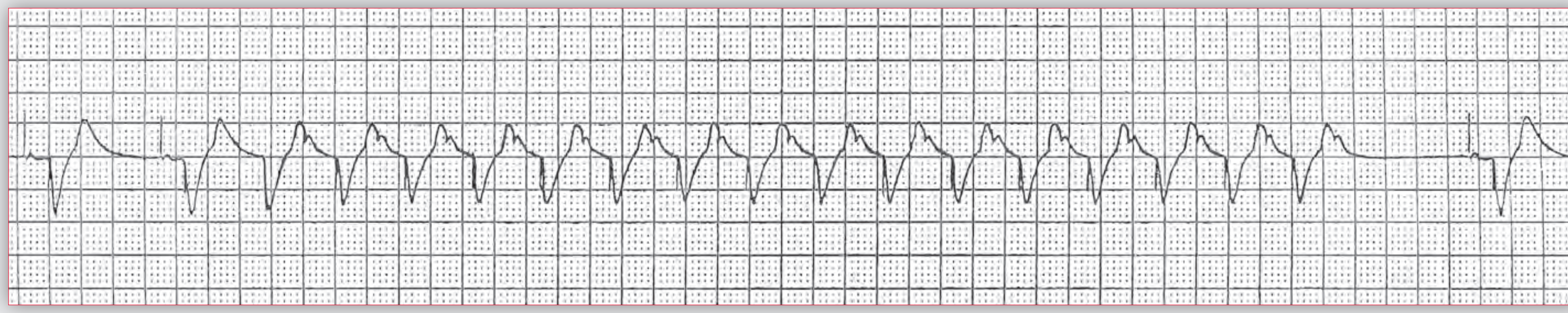
A pacemaker-mediated tachycardia is seen when there is a dual-chamber pacemaker (right atrium and right ventricle) and intact retrograde (VA) conduction back to the atrium via the AV node. In this situation, there are two pathways conducting impulses from the atrium to ventricle, *ie*, the pacemaker with an A and V lead and the normal AV node-His Purkinje system, as well as the potential for retrograde conduction. If a PVC produces retrograde atrial activation (VA conduction) that occurs at a time when it can be sensed by the atrial lead, the atrial impulse will result in ventricular activation, similar to what occurs with spontaneous atrial activity. If this paced ventricular complex again causes retrograde atrial activation, the process continues, resulting in a pacemaker-mediated tachycardia or what has also been called an endless-loop tachycardia. The pacing rate is at the upper rate limit of the pacemaker and is based on the ability of the atrial lead to sense the retrograde P wave, *ie*, the retrograde P wave occurs after the blanking period of the pacemaker, which is determined by the postventricular atrial refractory period (PVARP). When this happens, it can be terminated and prevented by placing a magnet over the pacemaker, eliminating all sensing, particularly atrial sensing. As the retrograde P wave can no longer be sensed, the tachycardia is terminated and prevented. Long term, this can be prevented by extending the PVARP, *ie*, increasing the blanking period or the duration of time during which the pacemaker is blind to or unable to sense atrial activity. ■

Notes

A 67-year-old man with a known sick sinus syndrome and a dual-chamber pacemaker implanted several years ago presents to the emergency department with complaints of intermittent palpitations that occur sporadically, lasting for seconds or as long as one hour. He is placed on telemetry in the emergency department, and while he is being evaluated he complains of a typical episode. The telemetry recording is presented.

What is the abnormality noted?

How can it be treated?



ECG 32 Analysis: Dual-chamber pacemaker,
AV sequential pacing, pacemaker-mediated tachycardia

The first two complexes on the rhythm strip have a pacing stimulus before the P wave (↑) as well as before the QRS complex (↓). Hence this is AV sequential pacing and the AV delay is 0.16 sec. There are no pacemaker stimuli associated with the third QRS complex (+) that is premature. The complex is wide and is a premature ventricular complex (PVC). Following this QRS complex, there is a notching on the downslope of the T wave (^); this is a retrograde P wave. The next QRS complex (fourth) (*) has a pacemaker stimulus before it (↓) and is followed by a retrograde P wave (^). Thereafter there is a repeating pattern of a paced QRS complex (↓) followed by a retrograde P wave (^). The interval between the retrograde P wave and the pacing stimulus is constant (⊓) (0.16 sec) and identical to the AV delay of the first two QRS complexes that are AV sequentially paced. Hence this is a pacemaker-mediated tachycardia, or endless-loop tachycardia, which is induced by a PVC that is associated with VA conduction. The rate is 140 bpm, which represents the upper rate limit of the pacemaker. The retrograde P wave is sensed by the atrial lead, resulting in a ventricular stimulus after the programmed AV delay of the pacemaker. The ventricular paced complex has VA conduction, and the process repeats. It can be seen that the tachycardia terminates after the 18th complex, which has a retrograde P wave (+) that is not sensed by the atrial lead and hence no ventricular stimulus occurs. This is due to the fact that the retrograde P wave occurs during the "blanking period," determined by the post-ventricular atrial refractory period (PVARP). During this time, atrial activity is not sensed and hence there is no ventricular stimulus. Upon termination there is a pause, followed by an AV sequentially paced complex (▲). The interval between the last ventricular paced stimulus, and the next atrial paced stimulus represents the lower pacing rate, *ie*, 52 bpm.

A pacemaker-mediated tachycardia can be acutely terminated by placing a magnet over the pacemaker, which inactivates all sensing. If the retrograde P wave is not sensed, it will not result in ventricular pacing. The pacemaker thus functions in a DOO mode, *ie*, it is fixed rate. To prevent this from occurring on a chronic basis, the PVARP of the pacemaker (blanking period) can be extended, *ie*, increasing the time during which an atrial impulse cannot be sensed. Since a pacemaker-mediated tachycardia is frequently initiated by PVC, many manufacturers have incorporated automatic PVARP extensions following a PVC. ■

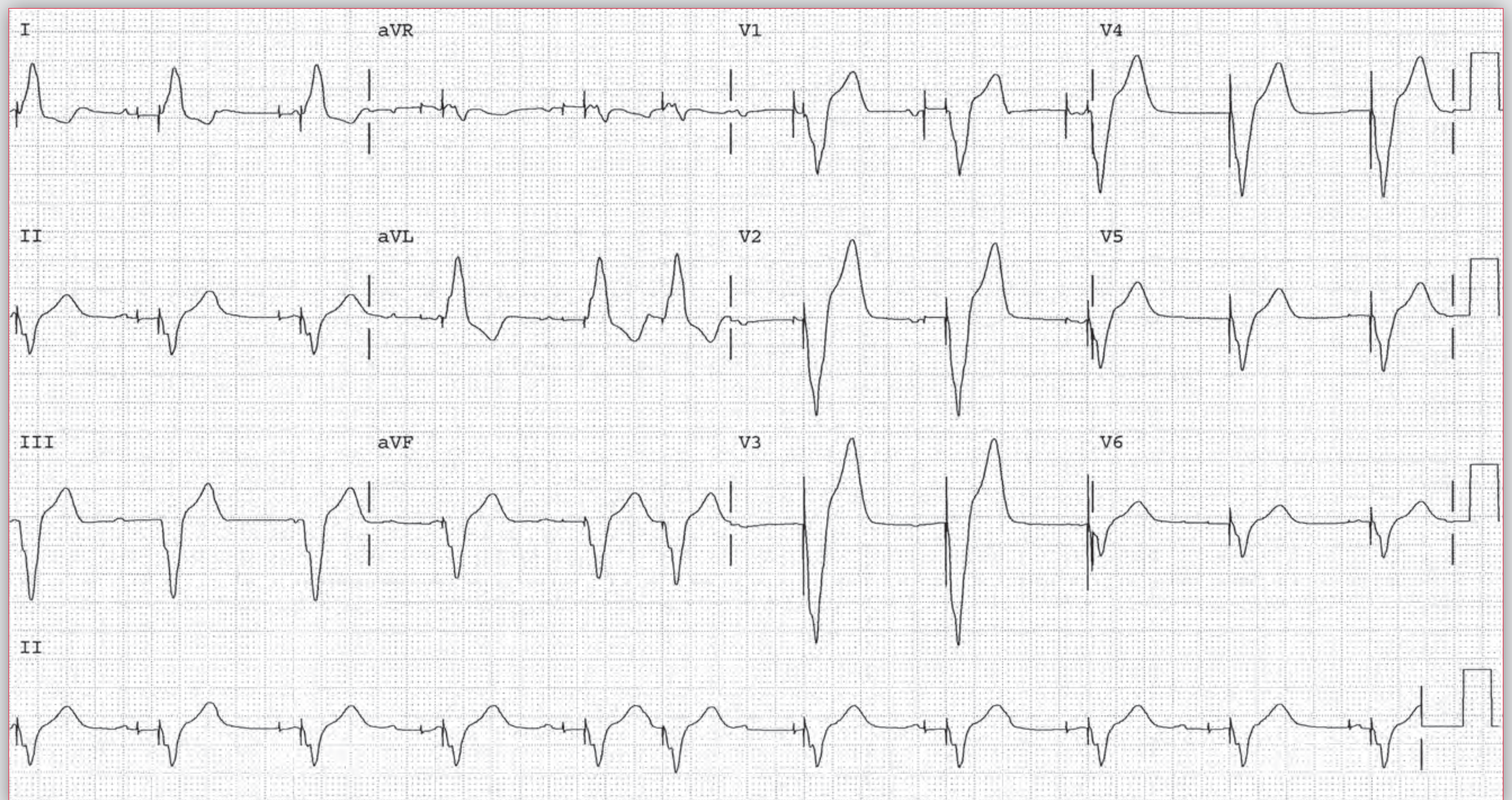
Notes

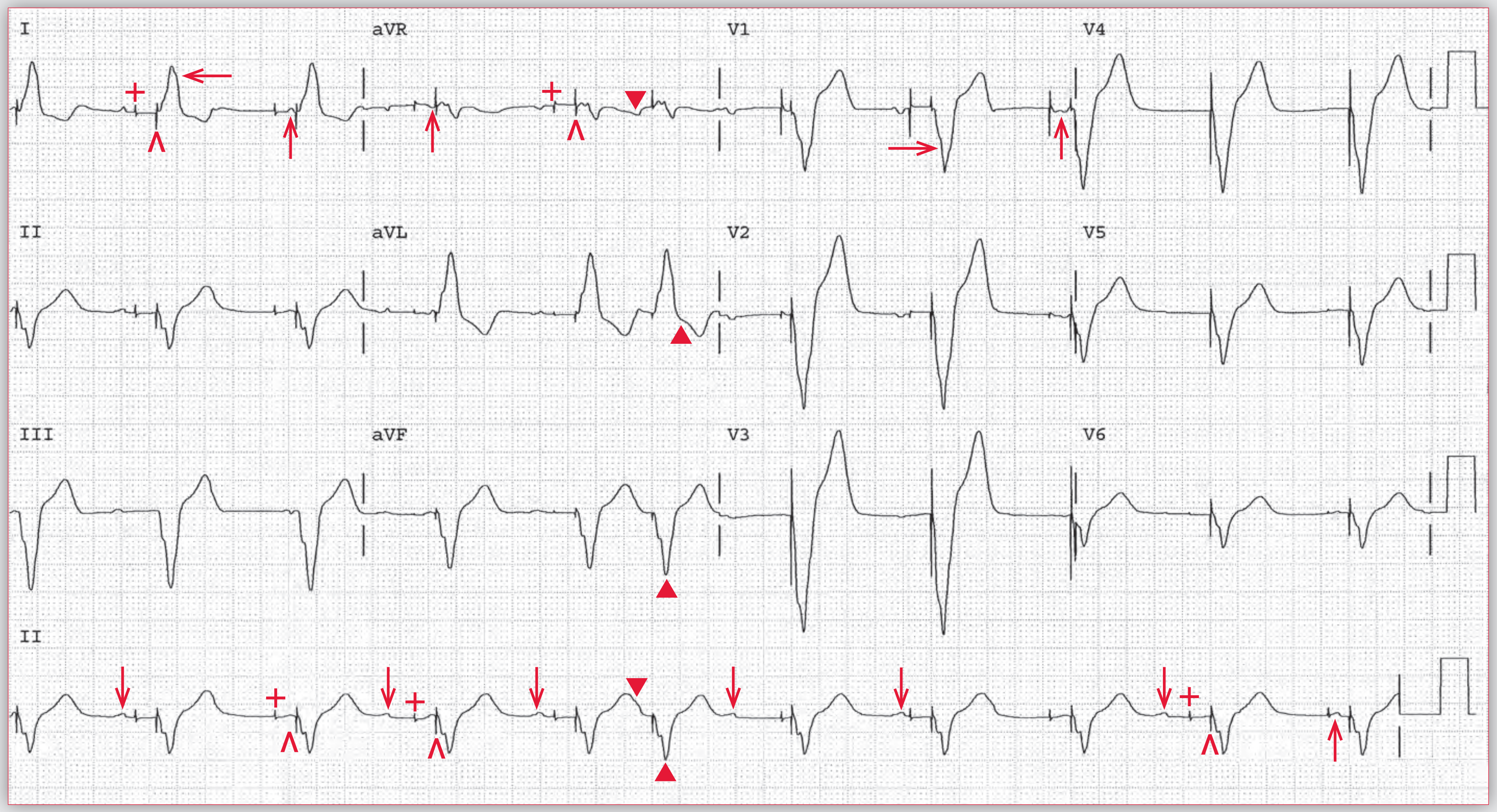
Core Case 33

An 80-year-old man with a history of a sick sinus syndrome and a pacemaker implanted 7 years ago is seen for a routine pacemaker check. He does not have any symptoms. An ECG is obtained, and the nurse practitioner is concerned that there is a pacemaker malfunction.

Do you agree that there is a malfunctioning pacemaker?

If so, what is the problem?





ECG 33 Analysis: Normal sinus rhythm, AV sequential pacing, failure of atrial sensing, premature atrial complex

There is a paced rhythm at a rate of 60 bpm. Two pacemaker stimuli can be seen before each QRS complex. The first is an atrial stimulus (+) while the second is the ventricular stimulus (^); the AV delay is 0.14 sec. The QRS complex duration is prolonged (0.16 sec), and it has a typical left bundle branch block morphology with a broad R wave in lead I (←) and a QS complex in lead V1 (→). Hence this is a right ventricular paced QRS complex. The QT/QTc intervals are prolonged (480/480 msec) but are normal when the prolonged QRS complex duration is considered (420/420 msec). There is a stable relationship between the ventricular stimulus and the paced QRS complex, and each QRS complex is preceded by a pacemaker stimulus. However, there is no regular relationship between the atrial stimulus and P waves (↓). Noted is a native P wave (↓) between the first and second, third and fourth, fourth and fifth, sixth and seventh, seventh and eighth, and ninth and tenth QRS complexes. The P wave is positive in leads I, II, and aVF and this is a normal sinus rhythm. There is no relationship between the P waves and the paced QRS complex; hence there is underlying complete heart block. Most of the P waves are followed by an atrial pacing stimulus. Hence there is failure of the atrial lead to sense the native atrial activity or P wave. When the atrial stimulus occurs shortly after the native P wave, the stimulus does not result in atrial activity, *ie*, there is no P wave in response to the atrial stimulus. This is due to the short interval between the P wave and the atrial stimulus; the atrial myocardium is still refractory and unable to respond to an impulse. However, if the atrial stimulus occurs at an appropriate time after the native atrial activity, it can capture the atrium and result in a paced P wave. This can be seen before the third, fourth, ninth, and last QRS complexes (↑). Therefore, there is problem with atrial sensing, but not atrial capture. However, the sixth QRS complex (▲) is premature and has only a ventricular paced stimulus before it. It appears that there is a P wave superimposed upon the T wave (▼) that is appropriately sensed, resulting in a ventricular output.

Although it is possible that the pacemaker is functioning in an asynchronous (nondemand or DOO mode), without any sensing and pacing independent of the underlying rhythm, this is not the case, as the sixth QRS complex (▲) is early and is in response to a premature atrial impulse (▼). Hence the atrial lead was capable of sensing this premature P wave, resulting in a ventricular stimulus after an appropriate AV delay. Another possibility is that the pacemaker is functioning in a DVI mode, *ie*, the pacemaker is incapable of sensing intrinsic atrial activity and will ignore any native P waves, *ie*, functional undersensing. However, this is not likely the case, as a premature atrial impulse was indeed sensed.

It appears that there is likely pacemaker malfunction and the right atrial lead is undersensing or not sensing atrial impulses. As the premature atrial impulse is indeed sensed, the failure to sense is intermittent, or may be related to the location of the atrial impulse in relation to the location of the right atrial lead, *ie*, an ectopic atrial impulse may originate closer to the atrial lead, resulting in appropriate sensing.

Therefore, there is evidence of pacemaker malfunction and the right atrial lead is undersensing or not sensing atrial impulses. The sensitivity of the right atrial lead can be increased. If ineffective, the right atrial lead could be replaced. Alternatively, the pacemaker mode can be changed to VVI. ■

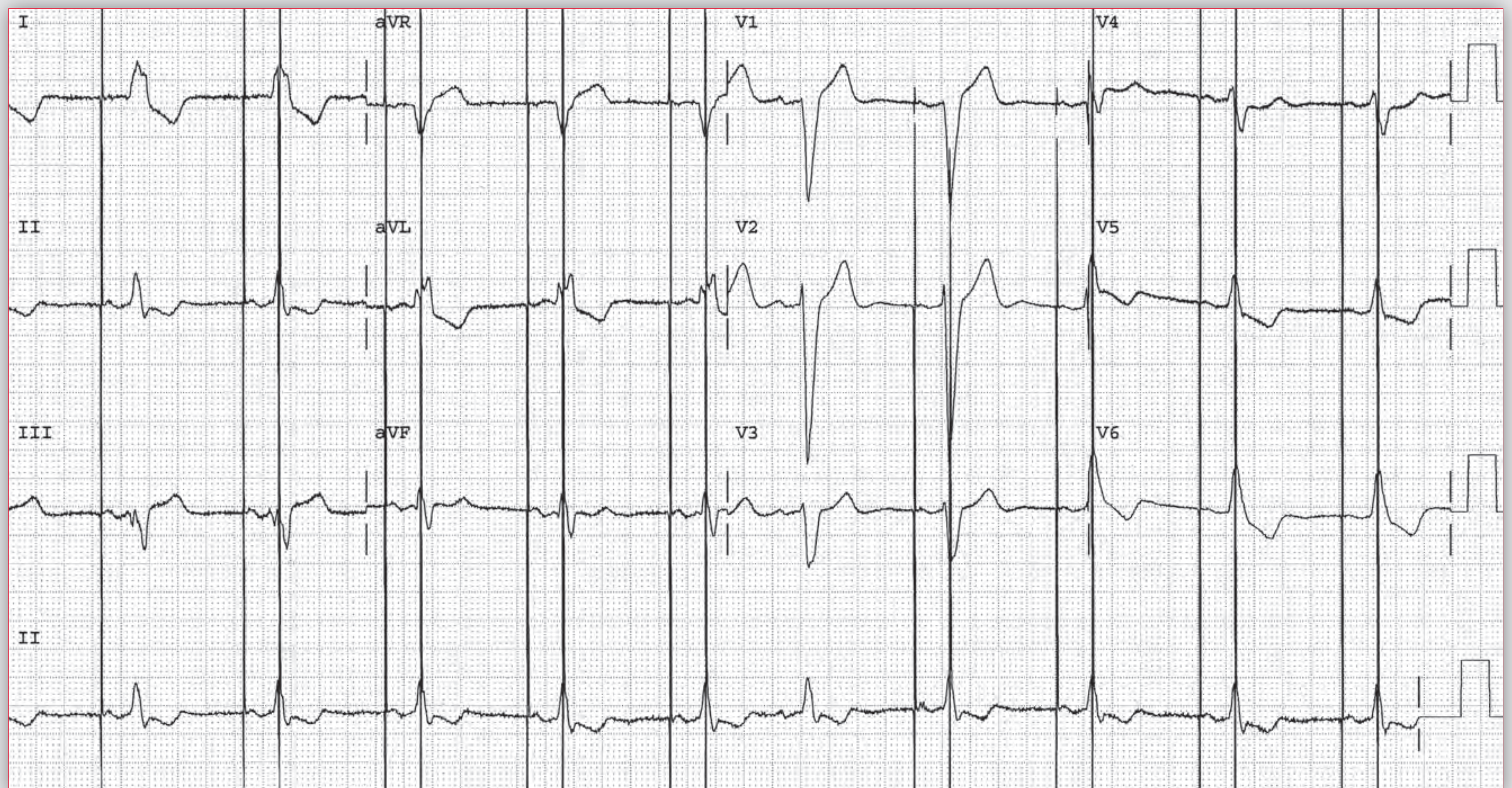
Notes

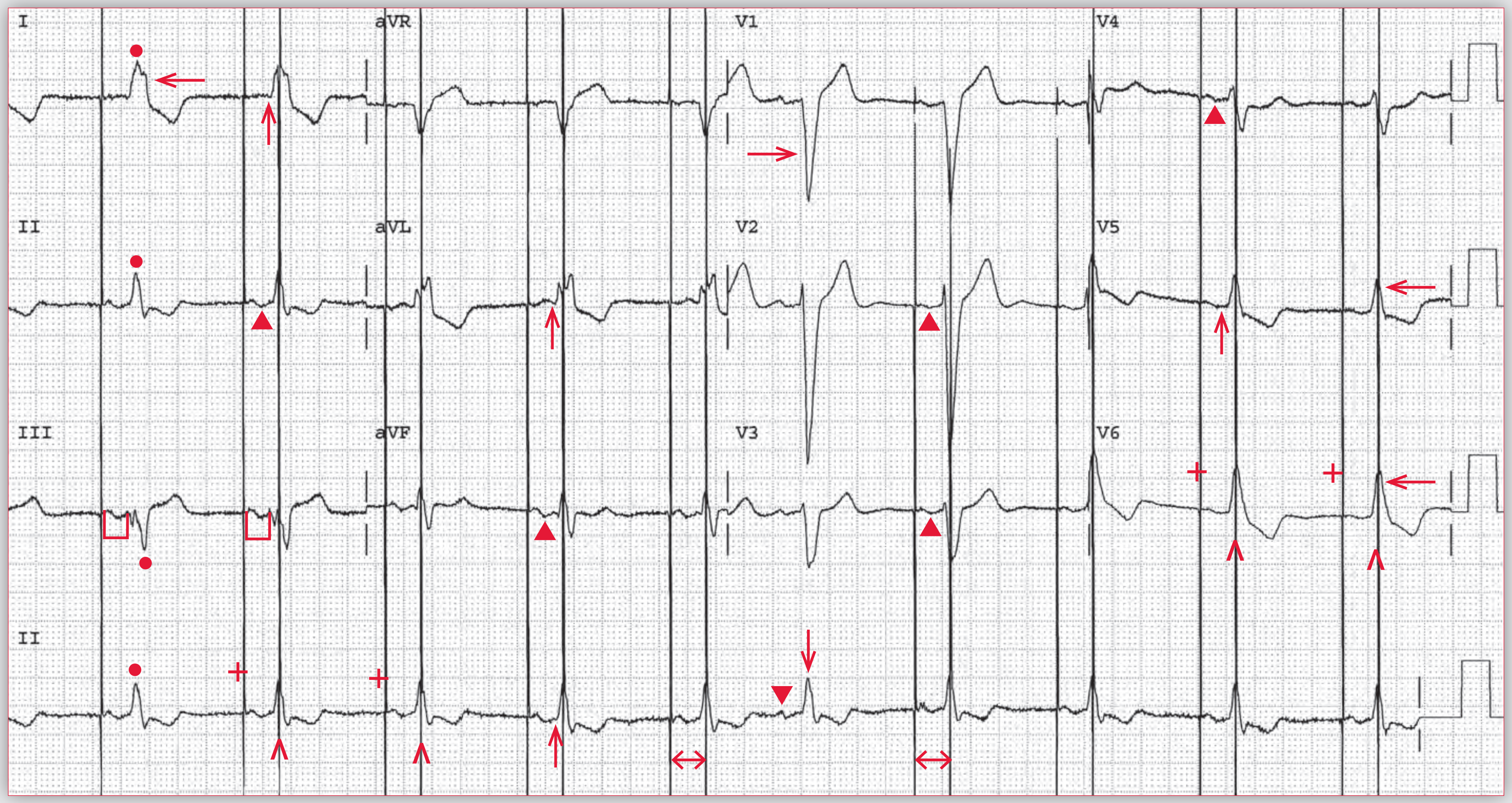
Core Case 34

A 74-year-old woman with a history of a sick sinus syndrome for which a pacemaker was inserted 7 years ago and a generator change one year ago presents for a routine pacemaker evaluation. An ECG is obtained, and it is felt that the pacemaker is functioning normally.

Is this a correct interpretation?

If not, what is the problem?





ECG 34 Analysis: Dual-chamber pacemaker, AV sequential pacing, failure of ventricular sensing. The native QRS complexes have a left bundle branch block morphology

There is a regular rhythm with evidence of two pacing stimuli, the first is atrial (+) and the second (^) is ventricular. The rate is stable at 60 bpm and each P wave (▲) (except for the sixth complex) is preceded by an atrial pacing stimulus. Hence there is an atrial paced rhythm. After each atrial stimulus, there is a ventricular stimulus (except for the first complex). The AV delay of the pacemaker is 0.24 sec (↔), while the native PR interval is 0.20 sec (⊔). It can be noted that the QRS complex begins prior to the ventricular pacemaker stimulus (↑). Therefore, the QRS complex is not a ventricular paced complex but is a native QRS complex. Hence the ventricular lead has failed to sense the native ventricular complex. The first QRS complex (●) demonstrates initial atrial pacing; however, there is no ventricular stimulus before the QRS complex, indicating that the failure to sense is intermittent. In addition, the sixth QRS complex (↓) is preceded by a P wave (▼) that is early and is without a preceding atrial pacing stimulus. The P-wave morphology is different from the paced P wave. This is either a normal sinus complex or a premature atrial complex (PAC). The QRS complex is the same as the others and is not preceded by a ventricular pacing stimulus. Hence the atrial lead appropriately sensed the PAC, and the ventricular lead has sensed the ventricular complex. This ECG shows that the ventricular electrode intermittently fails to sense.

The paced and nonpaced QRS complexes have the same morphology, confirming that the QRS complexes are not the result of the pacing stimulus but are the native QRS complexes. The duration of the QRS complex is increased (0.14 sec) and there is a morphology of a typical left bundle branch block with a broad R wave in leads I and V5–V6 (←) and a QS complex in lead V1 (→). The QT/QTc intervals are normal (420/420 msec and 380/380 msec when the prolonged QRS complex duration is considered).

The failure of the ventricular lead to appropriately sense may be a result of undersensing, which can be corrected by increasing the sensitivity of the ventricular lead. It may also be the result of a ventricular lead problem, in case it would need to be replaced. ■

Notes

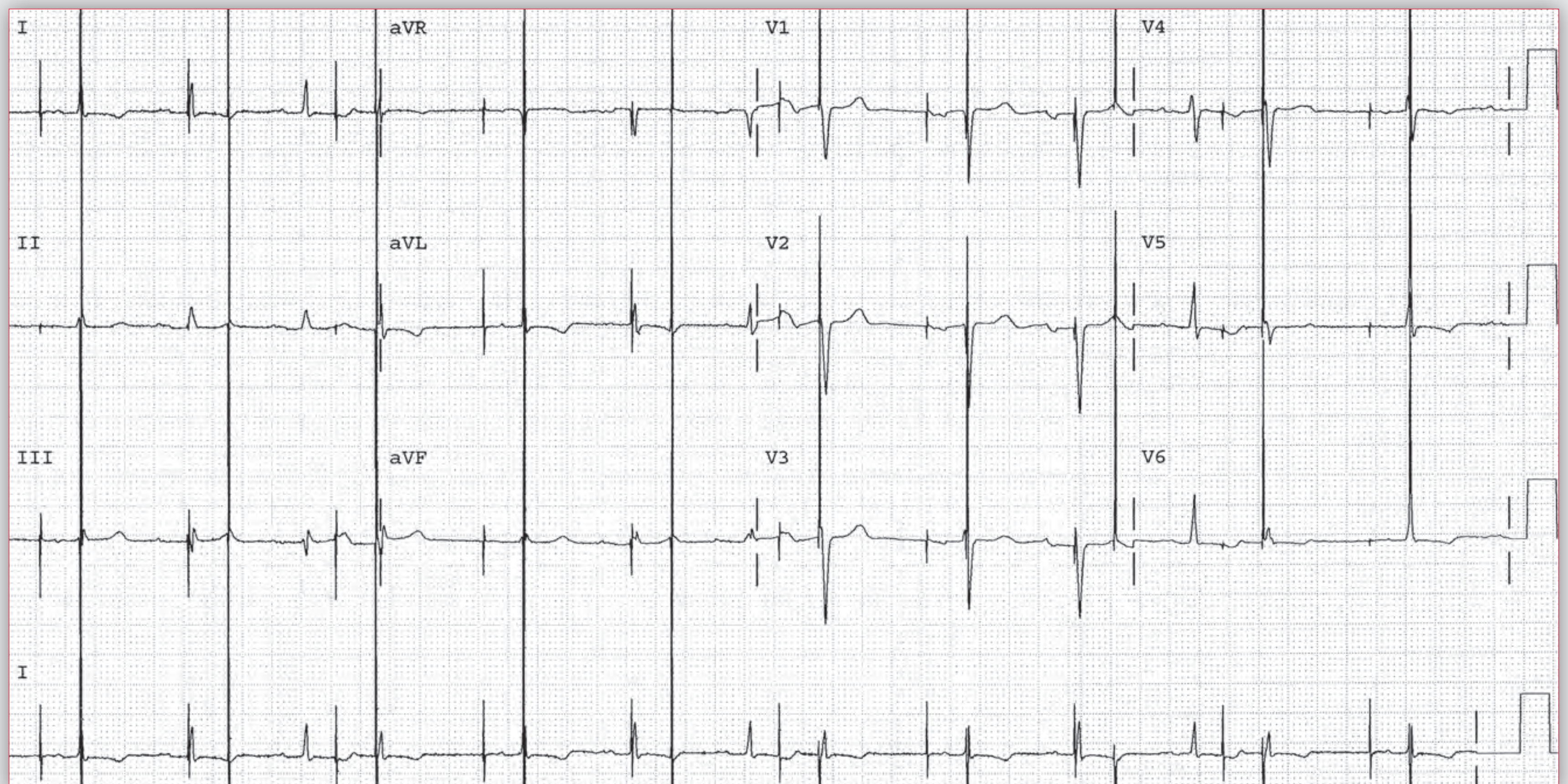
Core Case 35

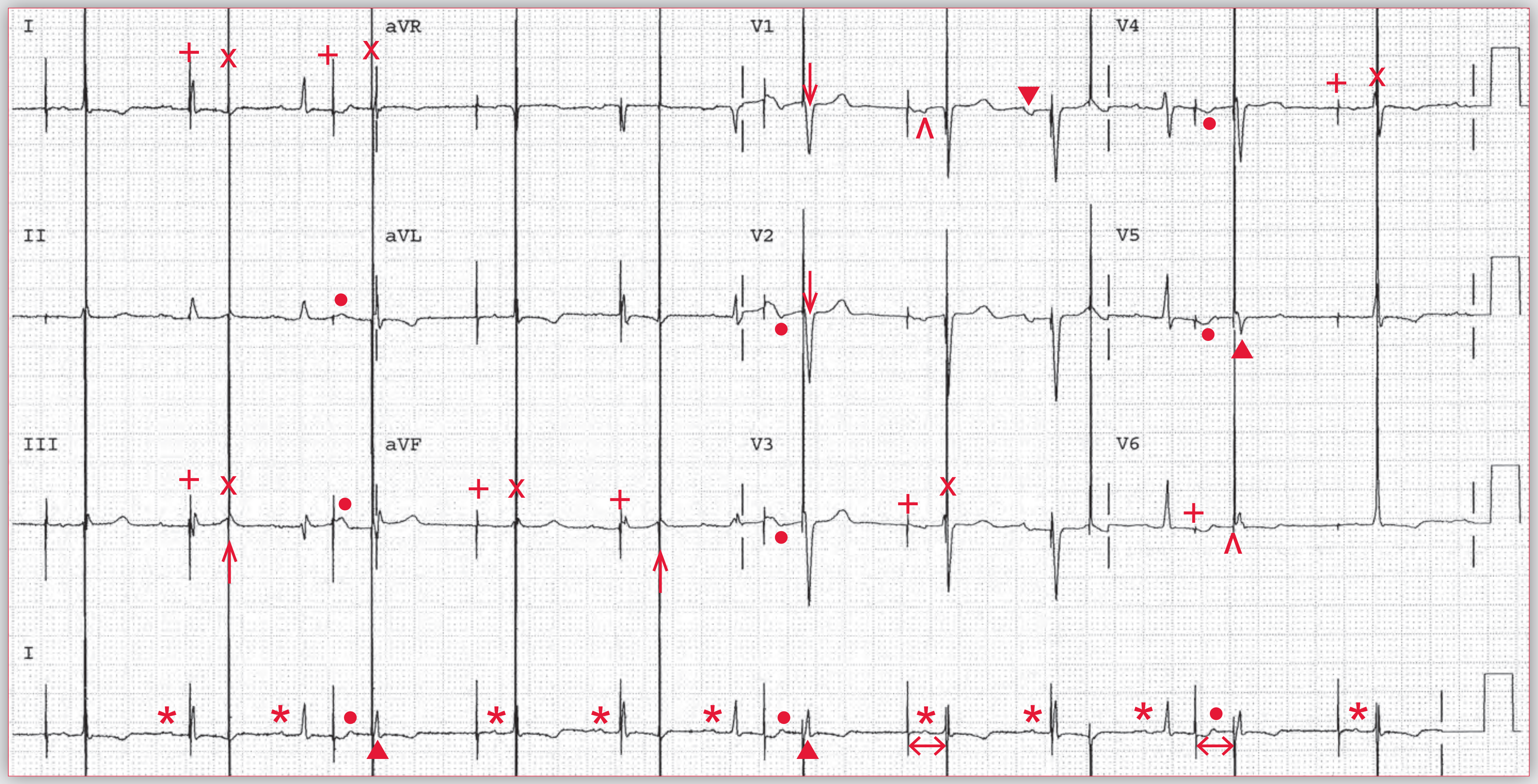
A 77-year-old man with a dual-chamber pacemaker is admitted to the hospital with an acute abdomen. Prior to urgent surgery a cardiology consult is obtained and the pacemaker is reprogrammed. After surgery, the patient is brought to the surgical intensive care unit, where a postoperative ECG is obtained.

Is the pacemaker function normal?

If not, what is the abnormality noted?

What therapy is appropriate?





ECG 35 Analysis: Normal sinus rhythm, low QRS complex voltage, failure of atrial and ventricular sensing, pacemaker functioning in a DOO mode, intermittent atrial capture

There is pacemaker activity seen, with a constant rate of 60 bpm. There are two pacing stimuli, atrial (+) followed by ventricular (x). The AV delay (↔) is 0.28 sec. The ventricular lead is not appropriately sensing spontaneous ventricular activity, nor is it capturing the ventricle, as the ventricular pacing stimulus has no relationship to the QRS complexes. The QRS complexes are narrow and normal in morphology. In some cases there is a pacemaker stimulus before the narrow QRS complex, and this might be the result of pseudofusion. However, this is not likely as each of the QRS complexes is preceded by a P wave (*) and the PR intervals is stable (0.20 sec). This is shorter than the AV delay of the pacemaker. Therefore, the QRS complexes appear to be the result of the spontaneous P wave. Also frequently seen is absence of atrial sensing and atrial capture, as the atrial pacemaker stimuli are most often unrelated to the P waves, which are native.

The QRS complexes are native complexes occurring at a regular rate of 76 bpm. Each QRS complex is preceded by a P wave (*) with a stable PR interval of 0.20 sec. The QRS complex duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QRS voltage in the limb leads is low (*ie*, < 5 mm in each lead). There appears to be a QS complex (↓) in leads V1–V2, suggesting an old anteroseptal myocardial infarction. The QT/QTc intervals are normal (340/410 msec).

Some of the atrial stimuli appear to be capturing the atrium. For example, the second complex in lead V1 has a P wave (^) after the atrial stimulus that has a different morphology from the P wave of the third complex in V1 that is a spontaneous P wave (▼), as there is no atrial pacemaker stimulus before it. In addition, there are some complexes that are early (▲) (fourth, eighth, and twelfth QRS complexes). They are preceded by an early P wave (●) that has a morphology that is different from the sinus P wave. In addition these P waves are preceded by an atrial stimulus, and they are paced P waves that result in a native QRS complex. Therefore, although the ventricular lead is not sensing the QRS complexes or capturing the ventricular myocardium (no relationship between pacing stimulus and native QRS complex which is narrow), there are occasional P waves resulting from the atrial stimulus. It can be seen that the atrial lead is able to capture the atrial myocardium when the atrial stimulus occurs at a time when the atrial myocardium is able to respond. None of the ventricular pacing stimuli are capturing the ventricle because the native QRS complexes cause the ventricular myocardium to be refractory.

Given the pattern that is seen, it is most likely that this represents asynchronous pacing (DOO), as the pacemaker is not inhibited by native atrial or ventricular activity, *ie*, it is not sensing spontaneous or native atrial or ventricular activity. However, it is possible that, in addition, there is failure of the ventricular lead to capture, although the fact that the pacing stimulus is simultaneous with the native QRS complex may represent pseudofusion. Other noncaptured ventricular stimuli occur after the QRS complex (↑), but the stimulus is not capturing, as the ventricular myocardium is still refractory. Hence it cannot be established if there is in fact failure to capture. It seems that prior to surgery, the pacemaker was programmed to a DOO mode so that the pacemaker would not be inhibited by external stimuli occurring during surgery. Postoperatively, the pacemaker should be reprogrammed to the mode that was present prior to surgery. ■

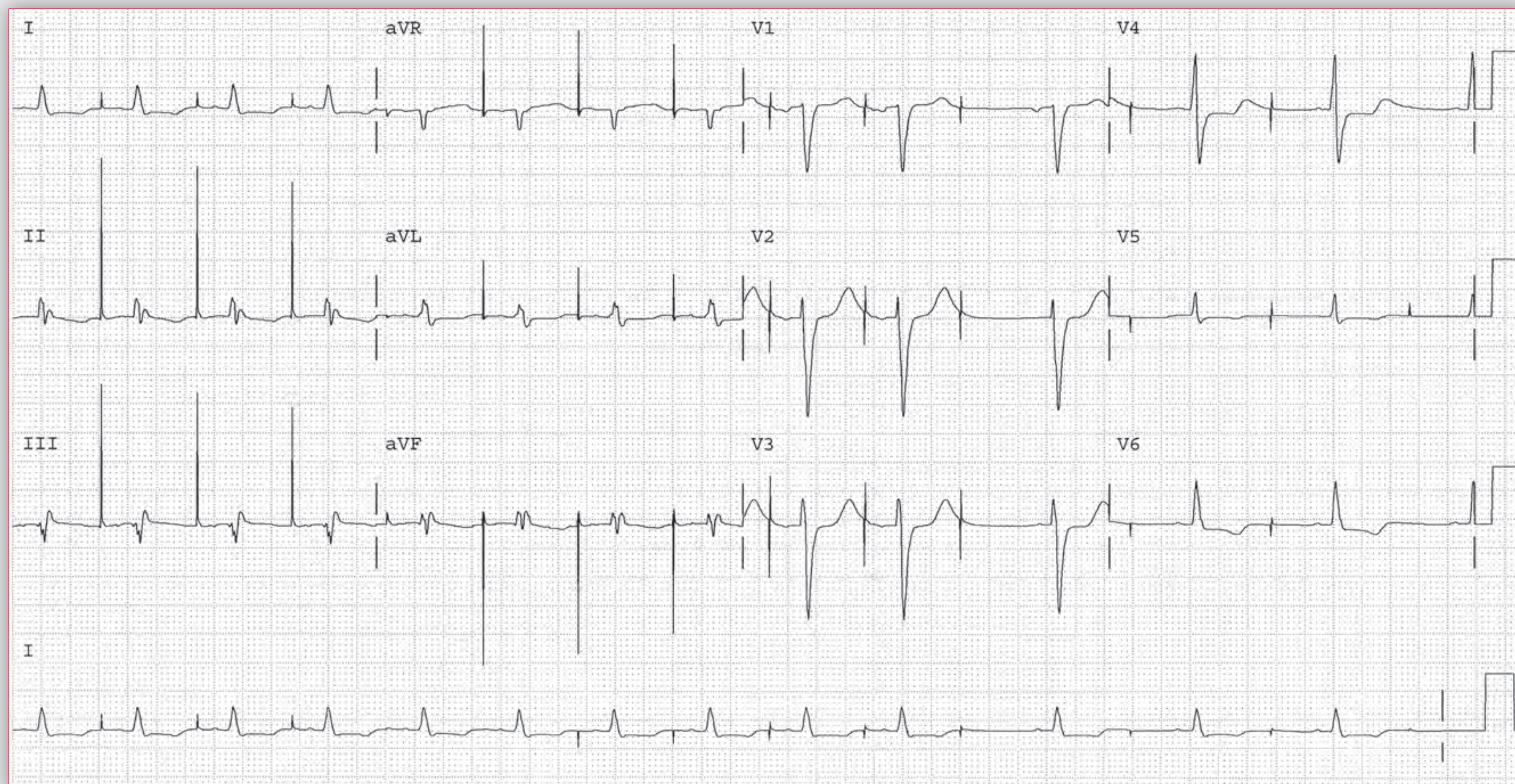
Notes

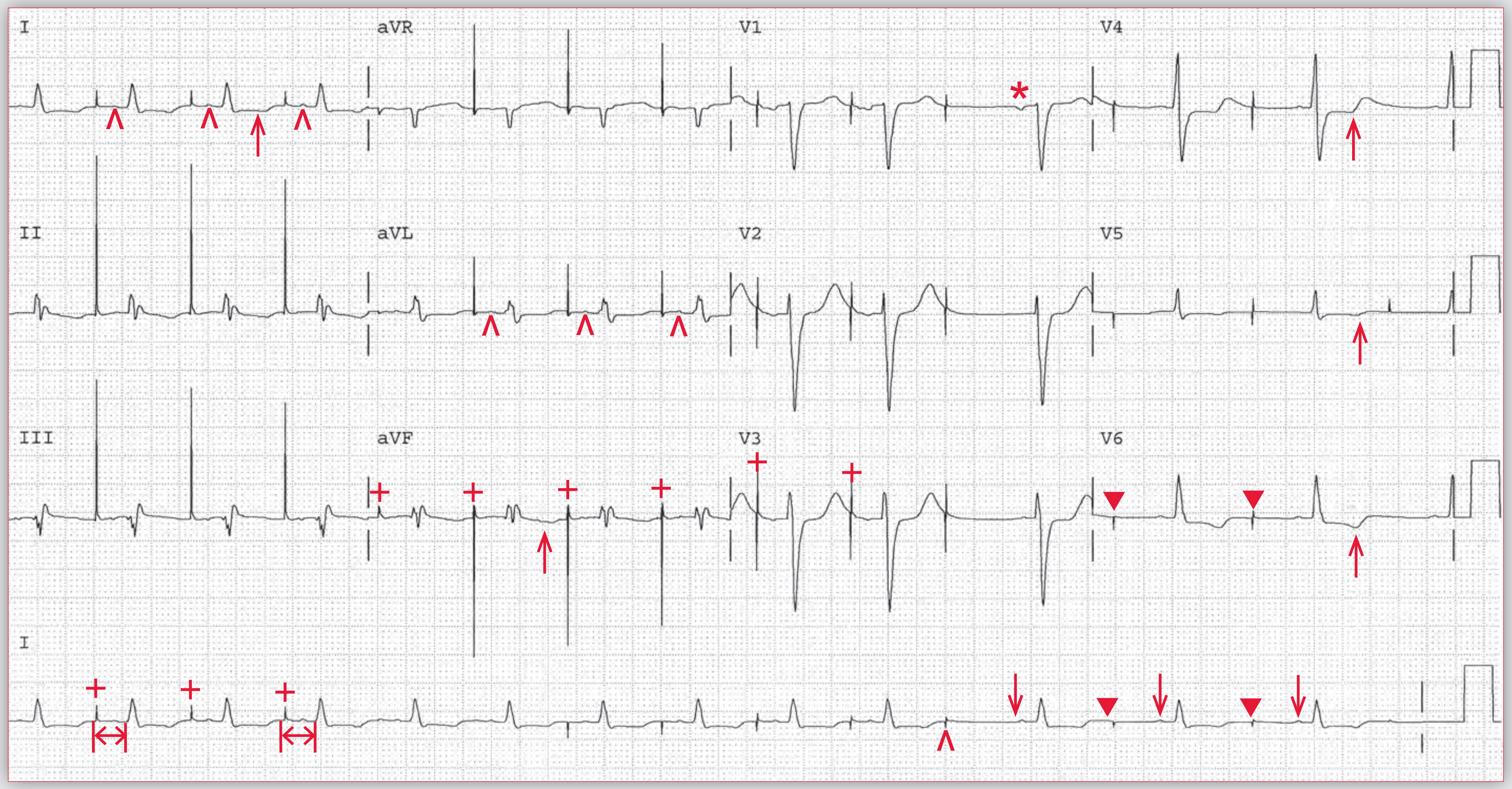
Core Case 36

A 65-year-old man with a history of a sick sinus syndrome resulting in a pacemaker implantation presents to his primary care provider for complaints of intermittent lightheadedness. This symptom is similar to what he experienced prior to receiving a pacemaker. His physical examination is normal. An ECG is obtained and was felt to show normal pacemaker function.

Is the pacemaker function normal?

If not, what is the abnormality noted?





ECG 36 Analysis: Atrial paced rhythm, intermittent failure of atrial capture, native QRS complexes have an intraventricular conduction delay and low voltage in the limb leads

The first part of the ECG shows a regular rhythm at a rate of 96 bpm. There is a pacemaker stimulus (+) seen before each P wave (^). The interval between the pacer stimulus and the QRS complex (*ie*, PR interval) is constant at 0.20 sec (↔). There is a native QRS complex as there is no pacer stimulus seen before this waveform. The QRS complex duration is prolonged (0.14 sec), and there is no pattern consistent with a bundle branch block. Therefore, this is an intraventricular conduction delay. There is low QRS voltage in the limb leads (< 5 mm in each lead) and nonspecific ST-T wave changes (↑). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (400/505 msec) but are only slightly prolonged when the widened QRS complex duration is considered (360/455 msec).

After the tenth QRS complex, the atrial stimulus (^) fails to capture the atrium as there is no P wave in response. As a result of failure to capture, there are native P waves (↓) at a rate of 60. Looking at V1, it can be seen that the native P wave (*) has a different morphology than the paced P wave. There are still atrial stimuli seen (▼), occurring at an irregular rate of 50-60 bpm. This is possibly due to the fact that the atrial stimulus is triggered by the ventricular complex, as the ventricular rate is also slower. Hence there is continued failure of the atrial lead to capture. This may be because the energy of the atrial output is too low to capture on a regular basis or there is problem with the lead.

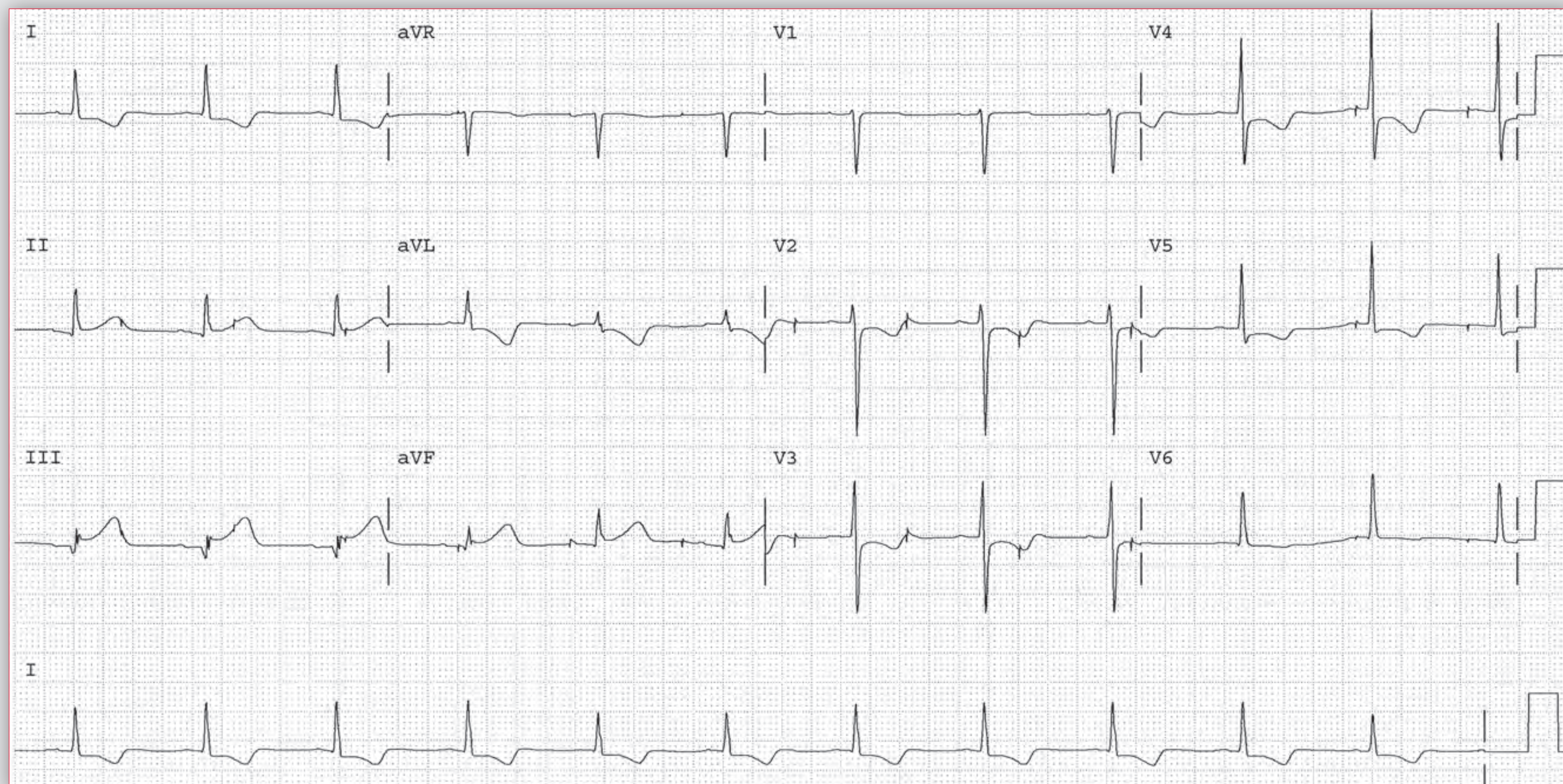
The underlying sinus rate is 60 bpm (when the pacemaker fails to capture) while the atrial pacing is at a faster rate. It is possible that the sinus rate is even slower than this, and without appropriate atrial capture, the heart rate (sinus rate) may have been even slower and associated with symptoms. ■

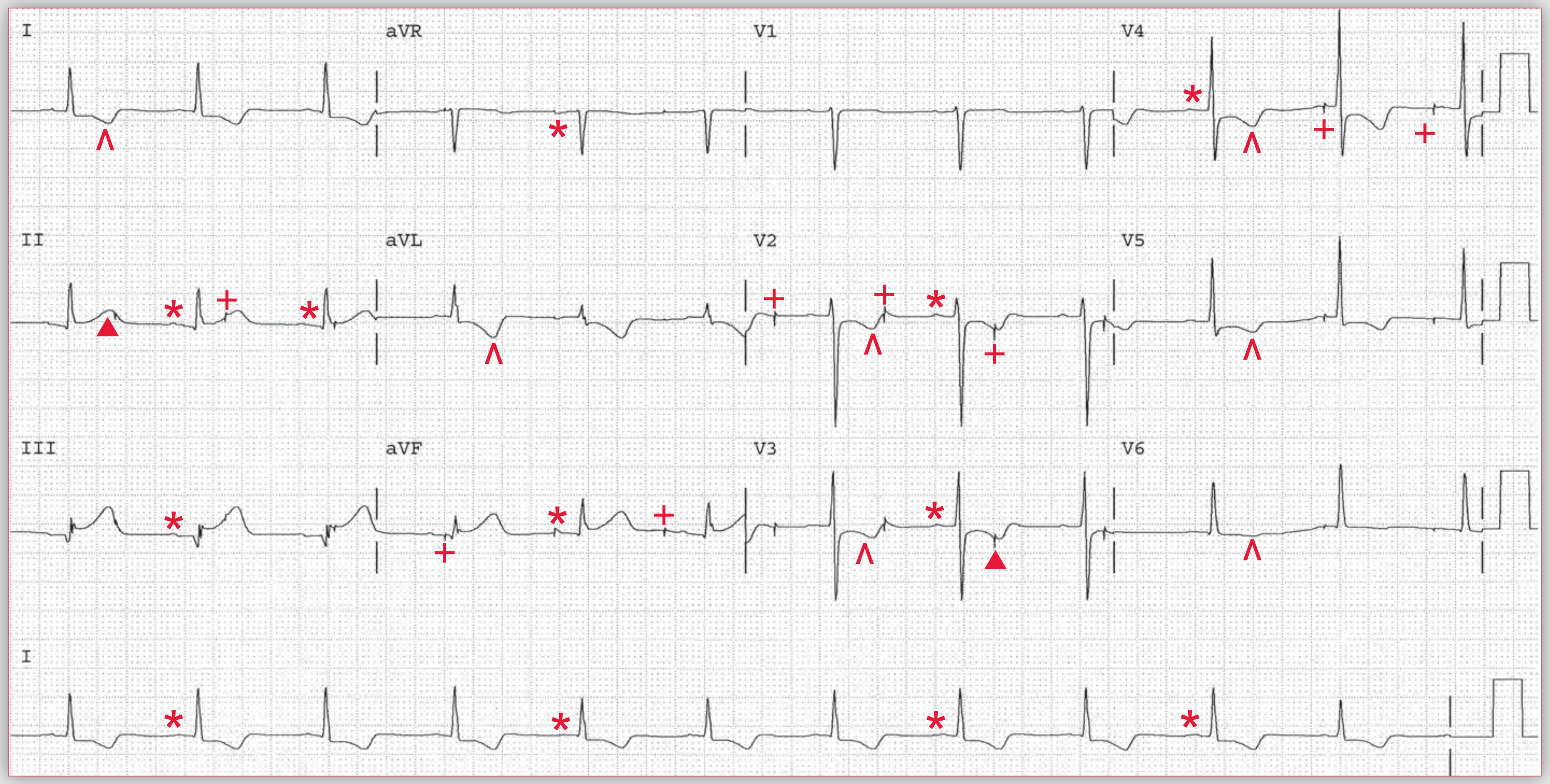
Notes

A 78-year-old man presents to the emergency department with complaints of intermittent lightheadedness associated with a feeling like he is going to pass out, although he denies actual syncope. He states that this has happened in the past and was the reason for a pacemaker implantation. An ECG is obtained and no abnormalities are detected.

What type of pacemaker does this patient have?

Is pacemaker function normal?





ECG 37 Analysis: Normal sinus rhythm, nonspecific ST-T wave changes, either atrial or ventricular pacemaker, failure to sense or capture

There is a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). Diffuse ST-T wave changes are seen in the lateral and anterolateral leads (^). The QT/QTc intervals are normal (400/425 msec).

Pacing stimuli (+) are seen occurring at a rate of 80 bpm. They are neither sensing nor capturing. However, it is not clear from the ECG if these are atrial or ventricular pacing stimuli. Therefore, the mode is either AOO or VOO. Although some of the pacemaker stimuli are occurring on the T wave (▲), the induction of a ventricular tachyarrhythmia (*ie*, ventricular fibrillation) is very unlikely as the amount of energy generated by the pacemaker is suboptimal (*ie*, it is less than the ventricular fibrillation threshold). However, in the presence of acute ischemia the ventricular fibrillation threshold decreases, and it is possible that the amount of energy generated by the pacemaker is adequate to provoke ventricular arrhythmia. ■

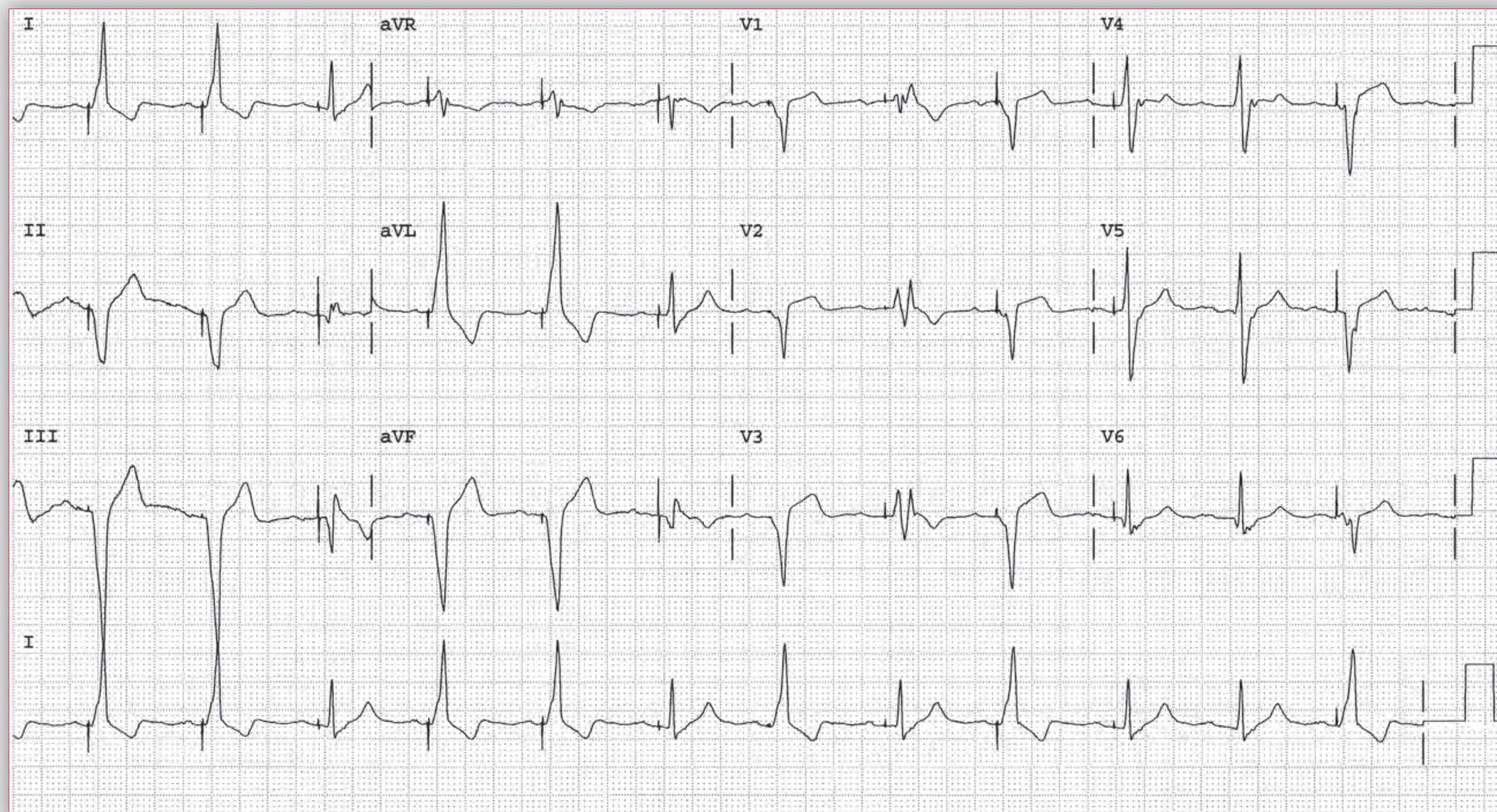
Notes

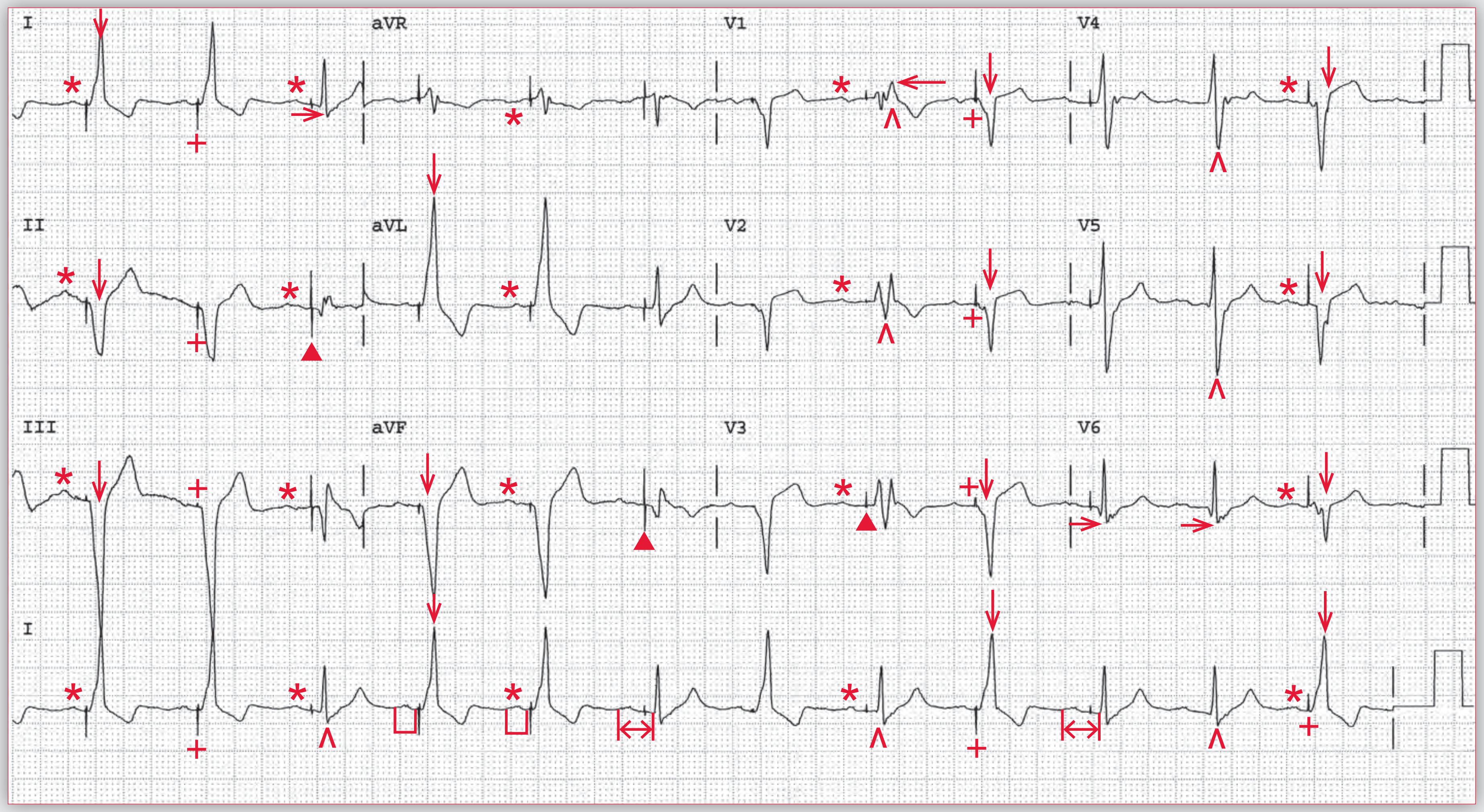
Core Case 38

An 85-year-old man who previously had a pacemaker inserted for unclear reasons, presents to the emergency department with complaints of chest discomfort that occurred while he was doing his usual walk in his neighborhood. The chest discomfort was across his chest and radiated to his neck and arm. The episode lasted about 45 minutes and although the discomfort had abated, he was still experiencing mild residual discomfort. In the emergency department his physical examination was normal and an ECG showed a paced rhythm at a rate of 76 bpm. His initial cardiac biomarkers were negative. He was admitted to the hospital for observation. Several hours later, he had a recurrent episode of chest discomfort and an ECG was obtained. The nurse was concerned and called the cardiologist.

What does the ECG show?

Is there any abnormality that is of concern?





ECG 38 Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous) mode, right ventricular pacing, intermittent failure of ventricular lead capture

There is a regular rhythm at a rate of 76 bpm. There is a P wave (*) before each QRS complex. Some of the QRS complexes are wide (0.14 sec) and have a left bundle branch block (LBBB) morphology (↓) with a broad R wave in lead V1 and a QS complex in lead V1. Others QRS complexes (third, sixth, eighth, tenth, and eleventh) are also wide (0.14 sec), but have a morphology typical of a right bundle branch block (RBBB; ^) with an RSR′ in V1 (←) and broad S wave in leads I and V4–V6 (→). There is a pacemaker stimulus (+) immediately before the QRS complexes that have a LBBB morphology. These QRS complexes are the result of an impulse from the right ventricular pacing electrode. Hence there is atrial sensed ventricular pacing (P-wave synchronous or activated ventricular pacing) with a constant PR interval (AV delay) of 0.16 sec (⊔).

The QRS complexes with a RBBB morphology also have a ventricular pacemaker stimulus preceding them (▲) with the same AV delay of 0.16 sec. However, there is no ventricular capture, as the QRS is not a result of the ventricular stimulus, *ie*, the QRS complex morphology is different from that of the paced complex. In addition, the PR interval (↔), which is constant, is longer (0.24 sec) than the AV delay of the pacemaker. Hence there is intermittent failure of the ventricular electrode to capture. Both QRS complexes have the same QT/QTc that is prolonged (420/470 msec) but is normal when the prolonged QRS duration is considered (380/430 msec).

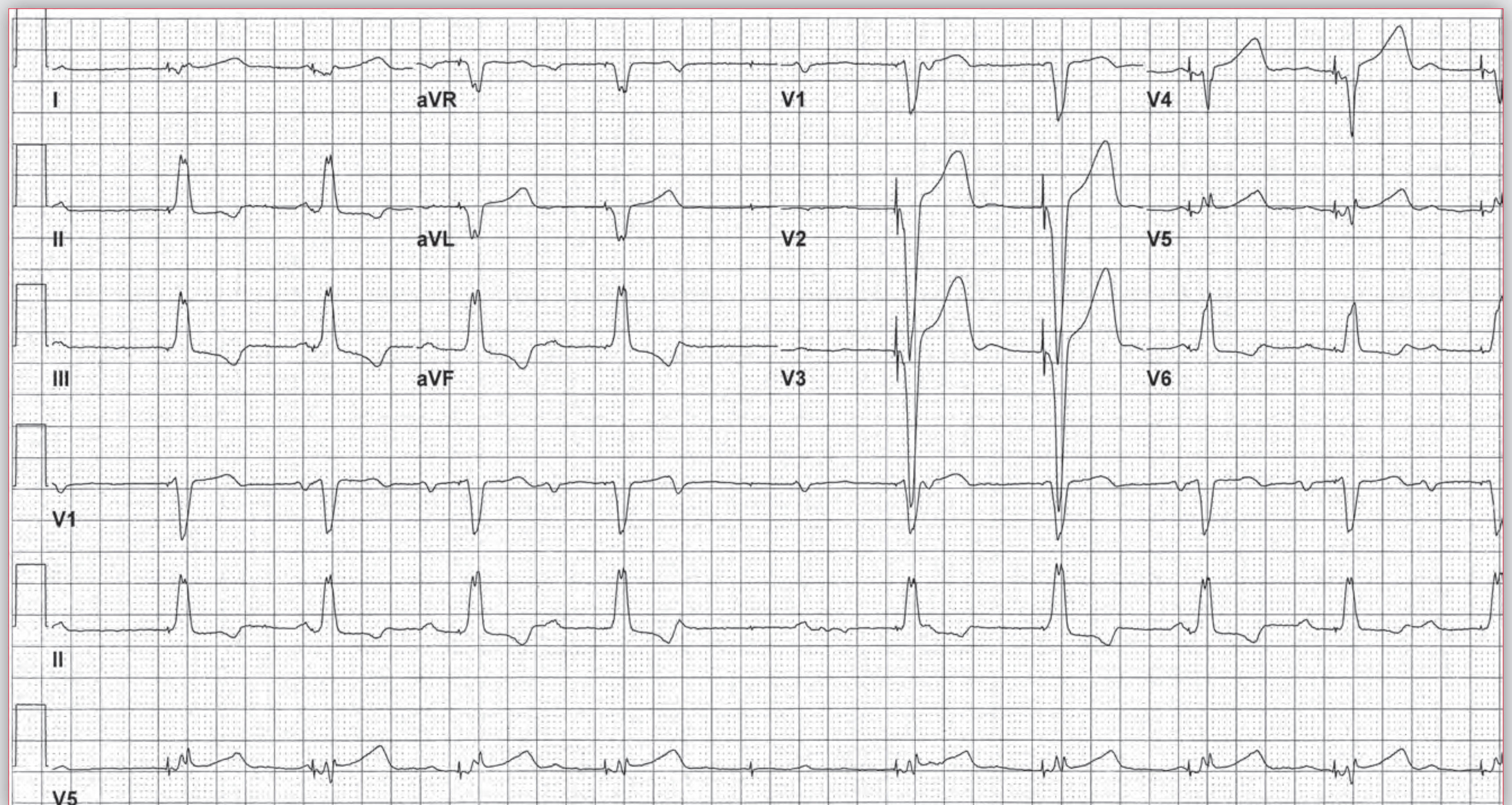
Although ST-segment changes cannot be interpreted with a paced QRS complex, as ventricular activation is not via the normal His-Purkinje system, ST-segment changes can be interpreted when the QRS complex is not the result of a pacemaker stimulus, *ie*, the complexes that have a RBBB. Even though there is a RBBB abnormalities of the left ventricle can be identified on the ECG, for example ischemia. In this case, there are no significant ST-segment changes associated with the episode of chest discomfort.

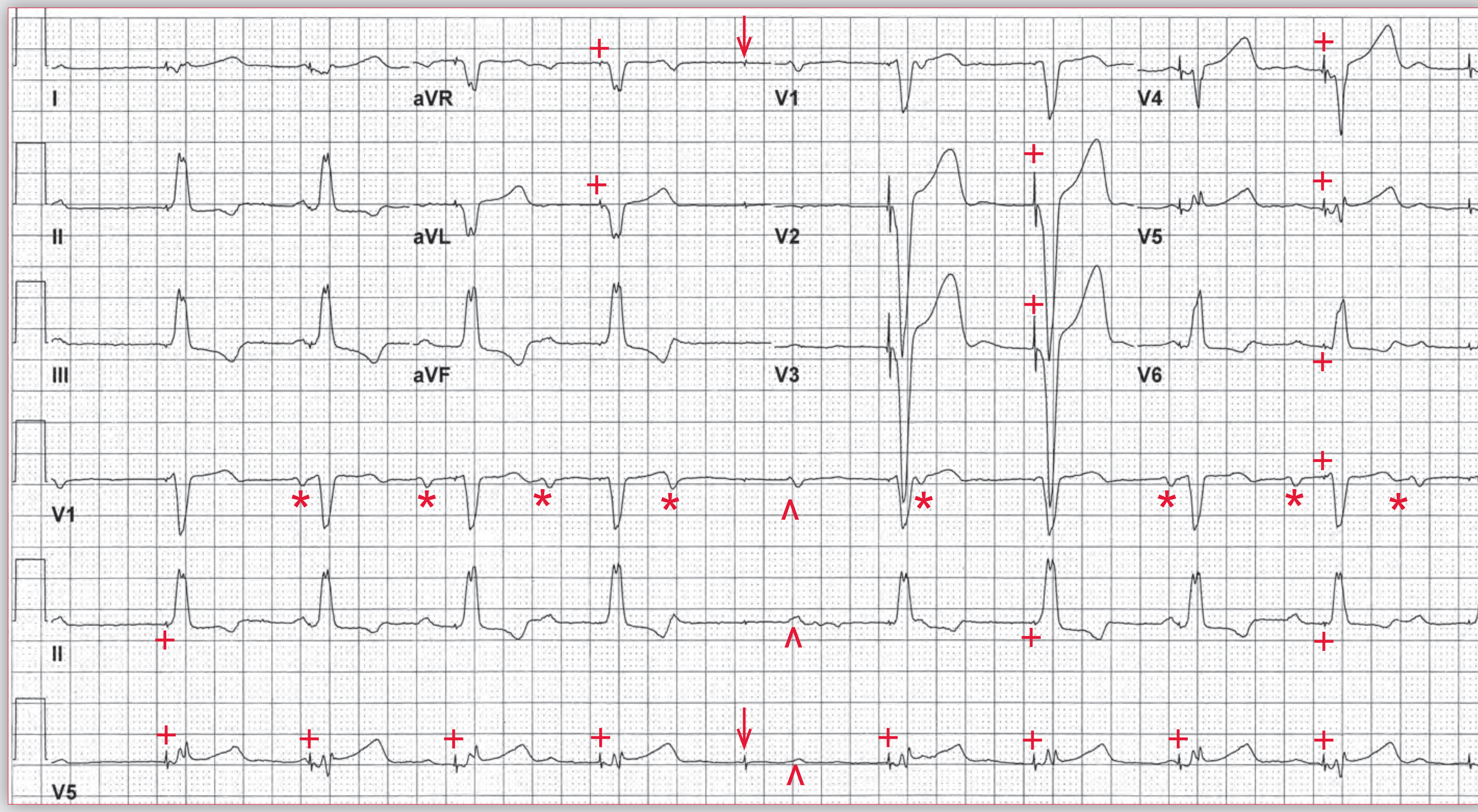
It is not clear if the intermittent failure to capture is a preexisting condition, reflecting a lead problem, or is new as a result of myocardial ischemia and a change in the right ventricular pacing threshold or energy necessary to reliably capture the ventricle. ■

Notes

Core Case 39

A 69-year-old man with a history of complete heart block requiring a pacemaker presents to the emergency department with complaints of intermittent lightheadedness.





ECG 39 Analysis: Normal sinus rhythm, complete heart block, VVI right ventricular pacing, intermittent failure to capture

There is a regular rhythm at a rate of 60 bpm. The QRS complex duration is increased (0.16 sec), and it has a left bundle branch block morphology. The QT/QTc intervals are prolonged (500/500 msec) but are normal when the prolonged QRS complex duration is considered (440/440 msec). There is a pacemaker stimulus (+) before each QRS complex. Hence this is a right ventricular paced rhythm. P waves can be seen (*) at a regular rate of 72 bpm. They are unrelated to the QRS complexes, *ie*, there is AV dissociation. As the atrial rate is faster than the paced rate, there is underlying complete heart block, and this is a VVI pacemaker.

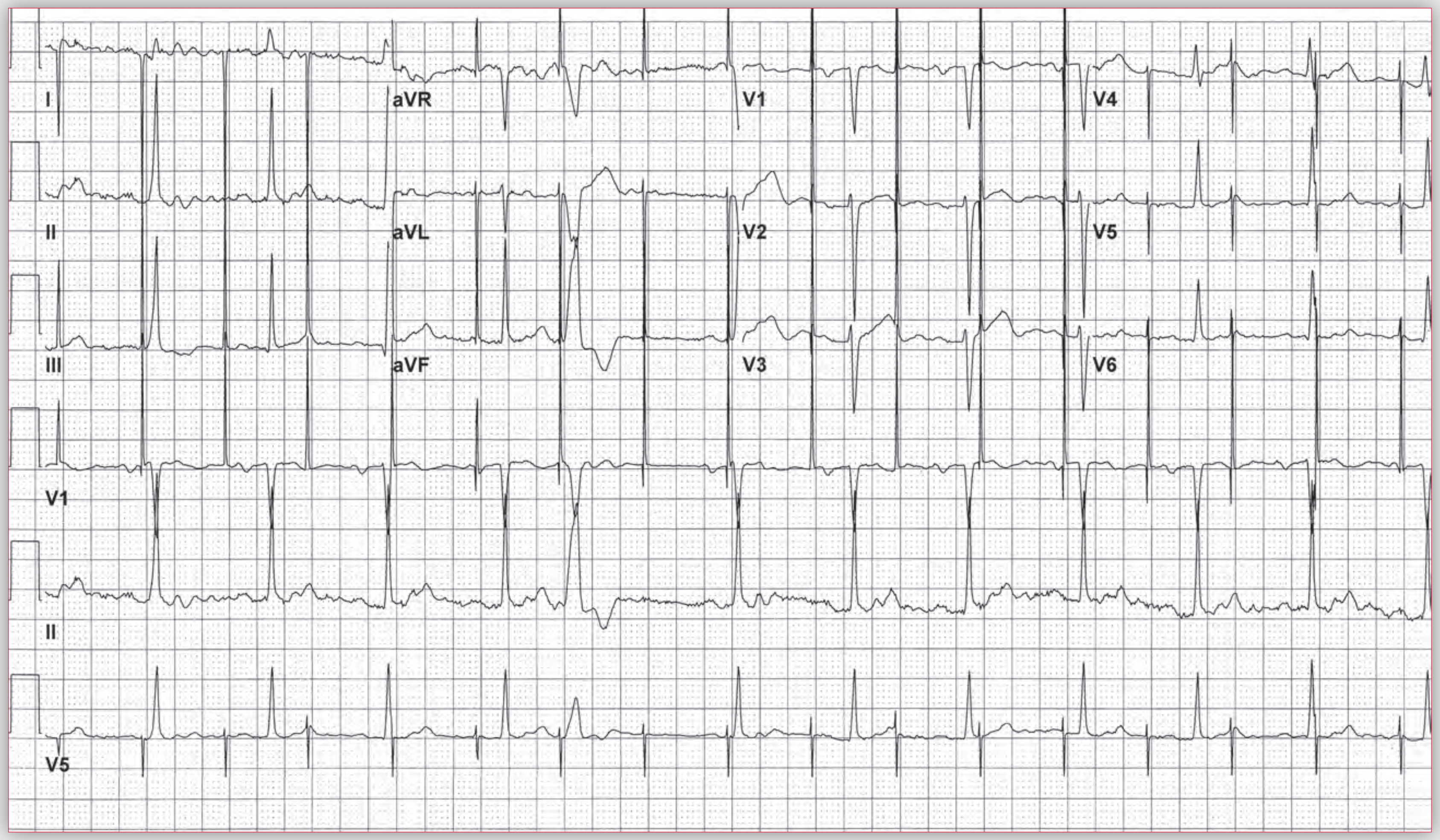
After the fourth QRS complex, there is a pause as a result of failure of an on-time ventricular stimulus to capture (↓), resulting in the absence of a paced ventricular complex. A nonconducted P wave (^) follows the pacemaker stimulus, and there is no QRS complex in response to the P wave, confirming complete heart block. Hence there is intermittent failure of ventricular capture.

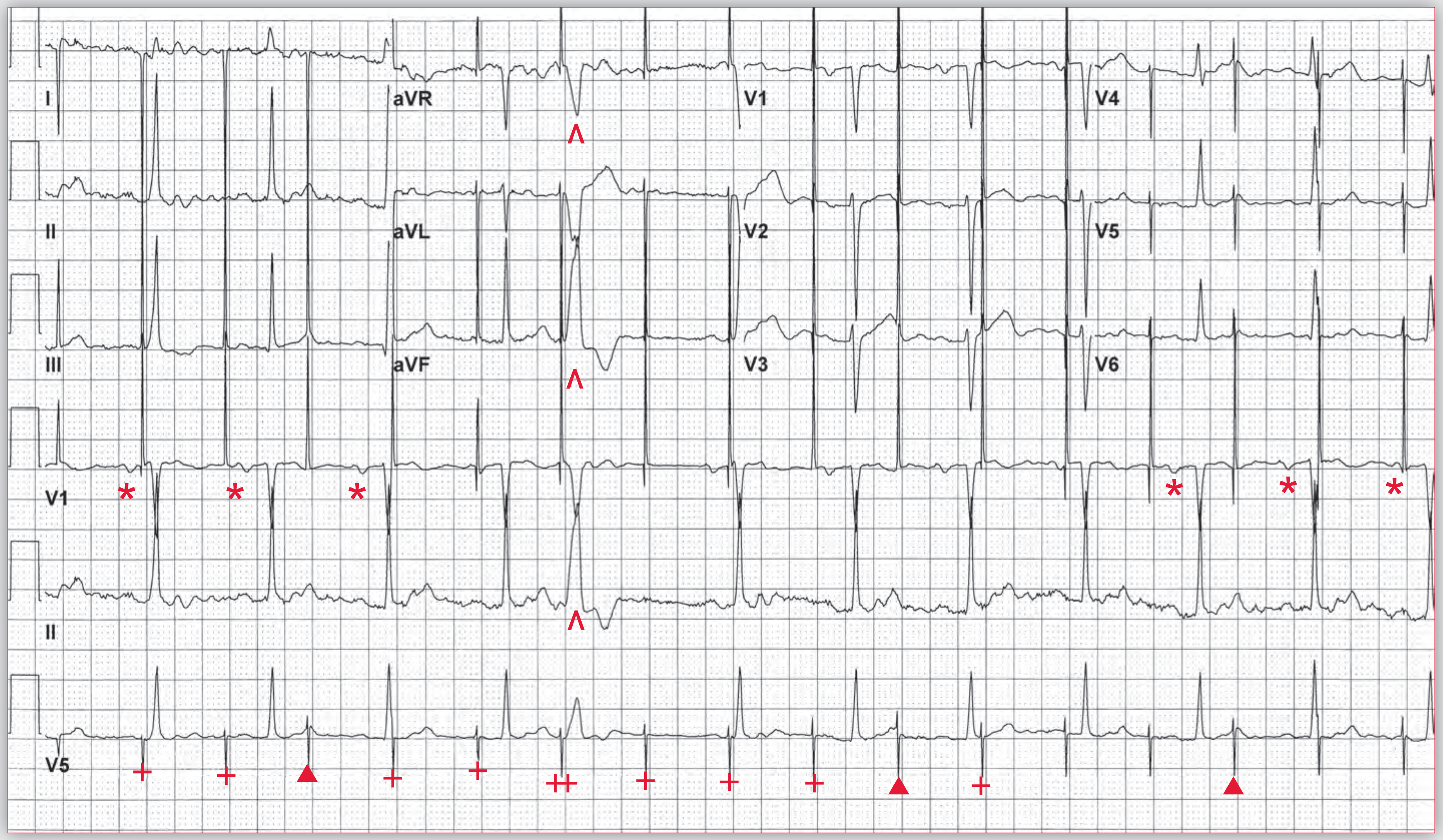
It is most likely that the symptoms are due to the failure of the pacemaker to capture in the presence of underlying complete heart block. It is not certain from this ECG is the complete heart block would be associated with an escape ventricular or junctional rhythm, as there is paced ventricular complex that ends the pause. ■

Notes

An 82-year-old woman is seen for a routine pacemaker evaluation. She does complain of episodic lightheadedness, but has no other symptoms. An evaluation of the pacemaker reveals an abnormality.

What does the ECG show and what is the problem with the pacemaker?





ECG 40 Analysis: Normal sinus rhythm, pacemaker failure to sense, pacemaker failure to capture

Pacemaker stimuli (+) can be seen at a rate of 100 bpm. There is an underlying native rhythm at a rate 76 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). Each QRS complex is preceded by a P wave (*) with a stable PR interval (0.24 sec), indicating a first-degree AV block or prolonged AV conduction. The QT/QTc intervals are normal (400/450 msec). There is no relationship between the pacing stimulus and the P waves or the QRS complexes. Therefore, there is failure of the pacemaker to sense or to capture. However, the fifth QRS complex (^) is early and has a prolonged duration. It appears to have responded to the pacing spike immediately preceding it (++). It is likely that this represents a single captured ventricular complex, although it is possible that it is a spontaneous premature ventricular complex that happens to occur simultaneously with the pacing stimulus.

The fact that the pacemaker rate is 100 bpm means that this is likely the result of a magnet that was placed over the pacemaker. This is commonly a magnet rate. It is unlikely that the lower rate of the pacemaker was set to 100 bpm. The magnet disables all sensing and hence the pacemaker functions in an asynchronous mode or VOO. This would account for the failure to sense, but does not account for the failure to capture. Appropriate capture would result in a ventricular paced rhythm at a rate of 100 bpm rather than a sinus rhythm at a rate of 76 bpm.

Although some of the pacemaker stimuli are occurring on the T wave (▲), the induction of a ventricular tachyarrhythmia (*ie*, ventricular fibrillation) is very unlikely as the amount of energy generated by the pacemaker is suboptimal (*ie*, it is less than the ventricular fibrillation threshold). However, in the presence of acute ischemia, the ventricular fibrillation threshold decreases, and it is possible that the amount of energy generated by the pacemaker is adequate to provoke ventricular arrhythmia.

As there is intermittent failure of the right ventricular pacing lead to capture, a new pacing lead is necessary. ■

Notes

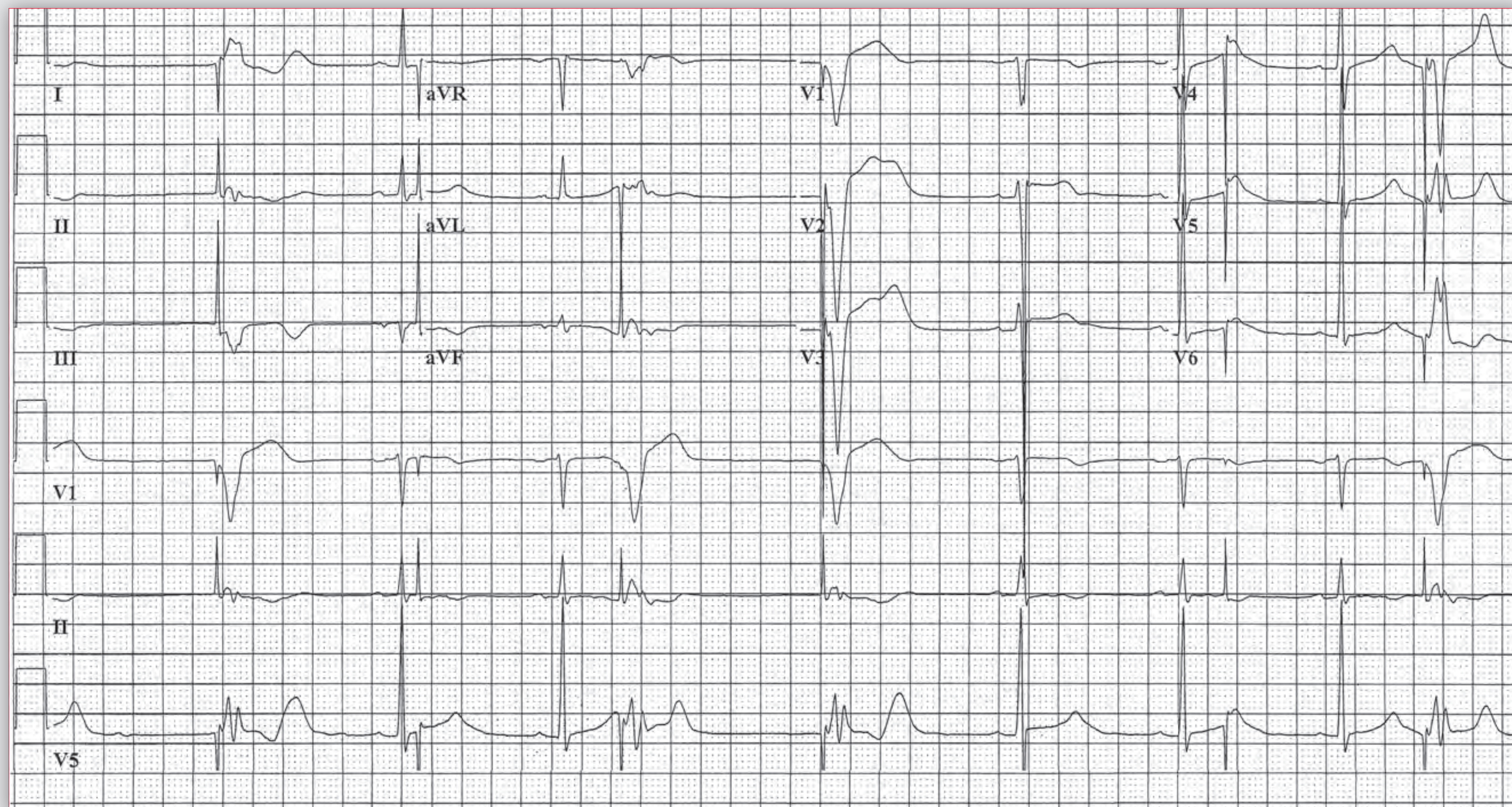
Core Case 41

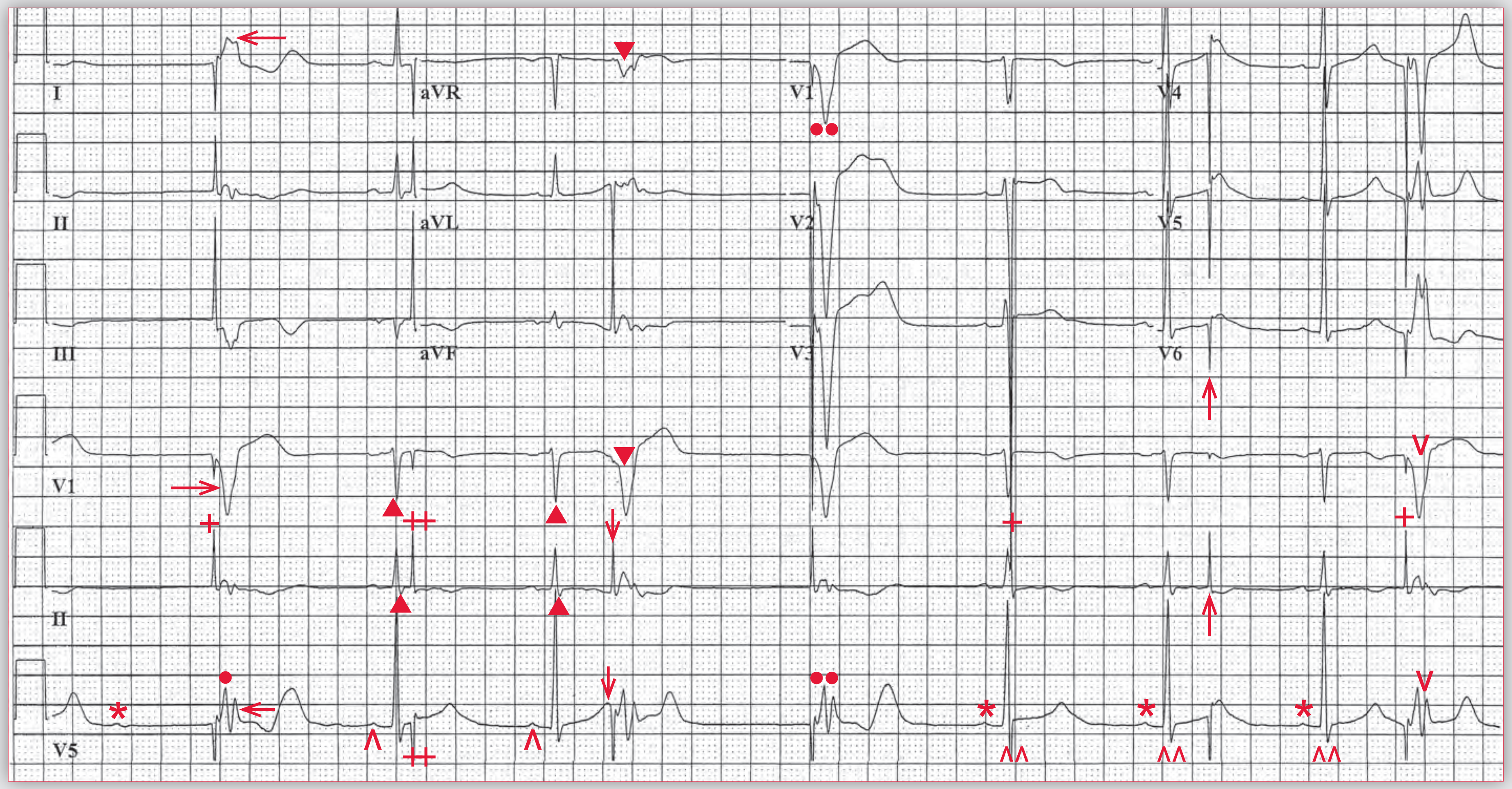
A 73-year-old woman presents to the hospital with acute cholecystitis and she is scheduled for an urgent cholecystectomy. The anesthesiologist obtains a history that she has a pacemaker for sick sinus syndrome, although the ECG shows a normal sinus rhythm. He asks the cardiologist to consult. The cardiologist reprograms the pacemaker prior to surgery. After surgery an ECG is obtained while the patient is in the recovery room. The nurse becomes concerned and stat pages the cardiologist.

What type of pacemaker does this patient have?

What is the abnormality noted?

Does this indicate pacemaker malfunction?





ECG 41 Analysis: **Normal sinus rhythm, VVI right ventricular pacemaker programmed to VOO, with failure to sense**

There is evidence of pacemaker activity, with ventricular stimuli (+) occurring in at a regular rate of 44 bpm. The rhythm is irregular, but with a pattern noted; hence it is regularly irregular. The first QRS complex (●), which is preceded by a pacemaker stimulus, is captured. The QRS complex has a left bundle branch block morphology with a broad R wave (←) in leads I and V5 and a QS complex in lead V1 (→). Prior to this complex, there is a nonconducted P wave (*), suggesting the presence of complete heart block resulting in a paced QRS complex. Hence it appears that this is a VVI pacemaker. The second and third QRS complexes (▲) have a normal duration (0.08 sec) and they are preceded by a P wave (^) with a PR interval of 0.18 sec; these are native sinus complexes at a rate of 56 bpm. The QT/QTc intervals are normal (440/425 msec). After the second QRS complex, there a pacemaker stimulus (++) within the ST segment, indicating a sensing failure. This stimulus fails to capture the ventricle as the myocardium is refractory (given the short interval between the native QRS complex and the pacing stimulus). There is a pacing stimulus (↓) shortly after the third QRS complex, which also represents failure to sense. However, this stimulus captures the ventricle, resulting in an early and wide QRS complex (▼) (ventricular paced) since the ventricular myocardium is not refractory as the interval between the native QRS complex and the pacing stimulus is longer. It is noted that the pacemaker stimulus does occur slightly after the apex of the T wave (*ie*, R on T). The fifth QRS complex (●●) is a result of the pacemaker stimulus and is a right ventricular paced QRS complex. There is no spontaneous P wave seen before this complex. The sixth, seventh, and eighth QRS complexes (^^) have a normal duration (0.08 sec) and are preceded by a P wave (*) with a stable PR interval (0.18 sec); they are normal sinus complexes. After the seventh complexes there is a pacemaker stimulus (↑) that fails to capture as it is early and the ventricular myocardium is still refractory. However, after the eighth QRS complex the ventricular stimulus does result in a paced QRS complex (v) as the interval between the sinus beat and pacemaker stimulus is longer and the ventricular myocardium has repolarized and is able to respond to the pacemaker stimulus.

Therefore, this ECG represents sinus rhythm, with episodic complete heart block. Although it appears that the patient has a ventricular pacing (VVI), there is failure of the ventricular electrode to sense the native QRS complex. Ventricular capture is intact and appropriate. The failure to sense may be the result of a problem with the right ventricular pacing lead or may be due to the fact that the pacemaker is functioning in a VOO mode with sensing disabled. This is the likely problem as the pacemaker was reprogrammed prior to surgery, from a VVI to a VOO mode so that electrical impulses resulting from surgery will not cause inappropriate suppression or inhibition of the pacemaker output. This is particularly important in a patient who is pacemaker dependent.

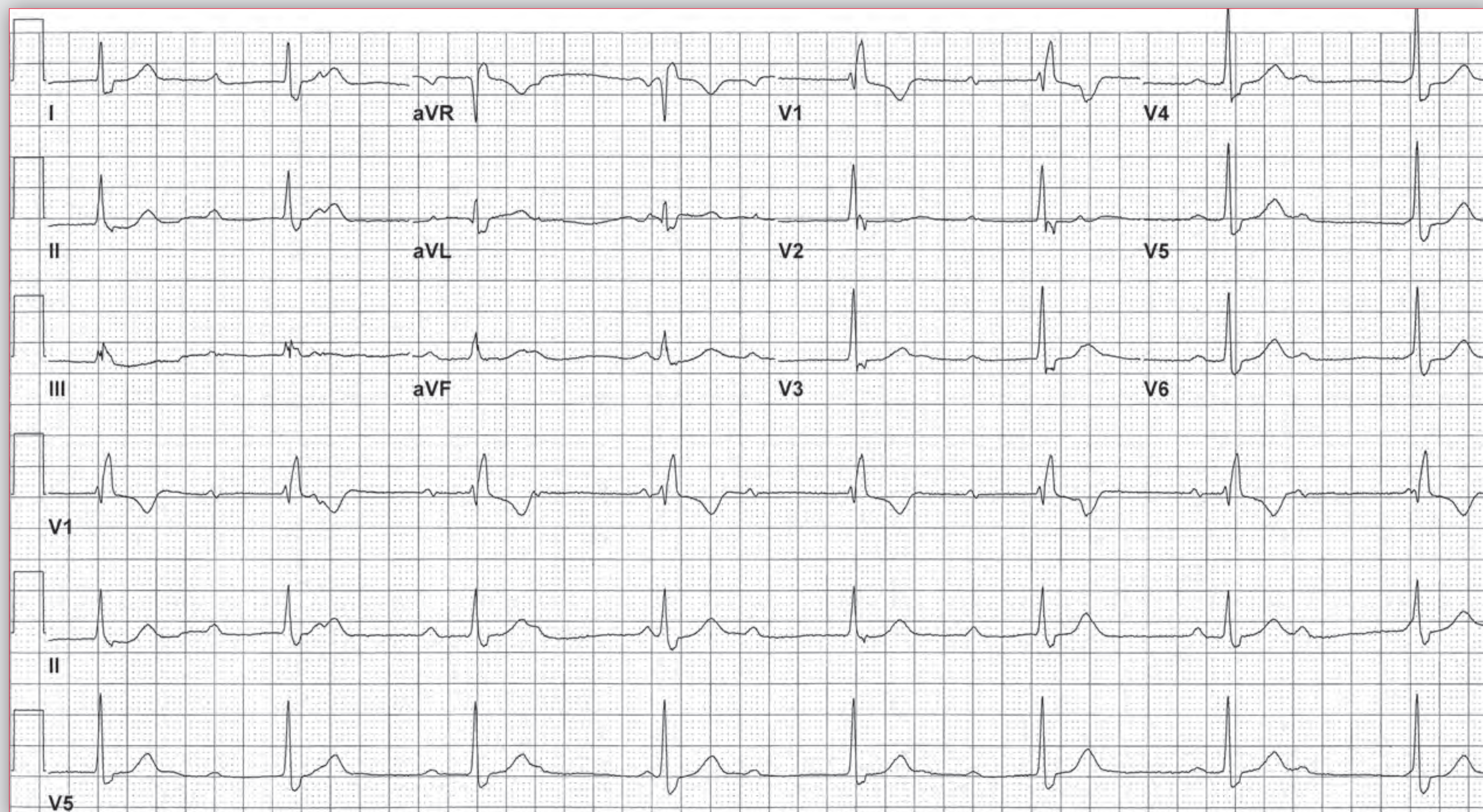
Although some of the pacemaker stimuli are occurring on the T wave (↓), the induction of a ventricular tachyarrhythmia (*ie*, ventricular fibrillation) is very unlikely as the amount of energy generated by the pacemaker is suboptimal (*ie*, it is less than the ventricular fibrillation threshold). However, in the presence of acute ischemia, the ventricular fibrillation threshold decreases, and it is possible that the amount of energy generated by the pacemaker is adequate to provoke ventricular arrhythmia. ■

Core Case 42

A 26-year-old male presents to his primary doctor with complaints of shortness of breath with everyday activities. Gradually over the past few weeks, he noticed he was no longer able to keep up with his friends during recreational basketball. Most recently, climbing the two flights of stairs at his home resulted in marked dyspnea.

He denies palpitations or lightheadedness. He denies fatigue, changes in appetite, bowel habits, or skin, hair or nail character. He denies signs of occult bleeding. He denies recent fevers. He does mention a recent illness that occurred shortly after a hiking trip to New England when he was febrile and noticed large, round red rashes that were

ECG 42A



associated with myalgias and arthralgias, but all symptoms resolved within two weeks. He did not seek medical attention at that time.

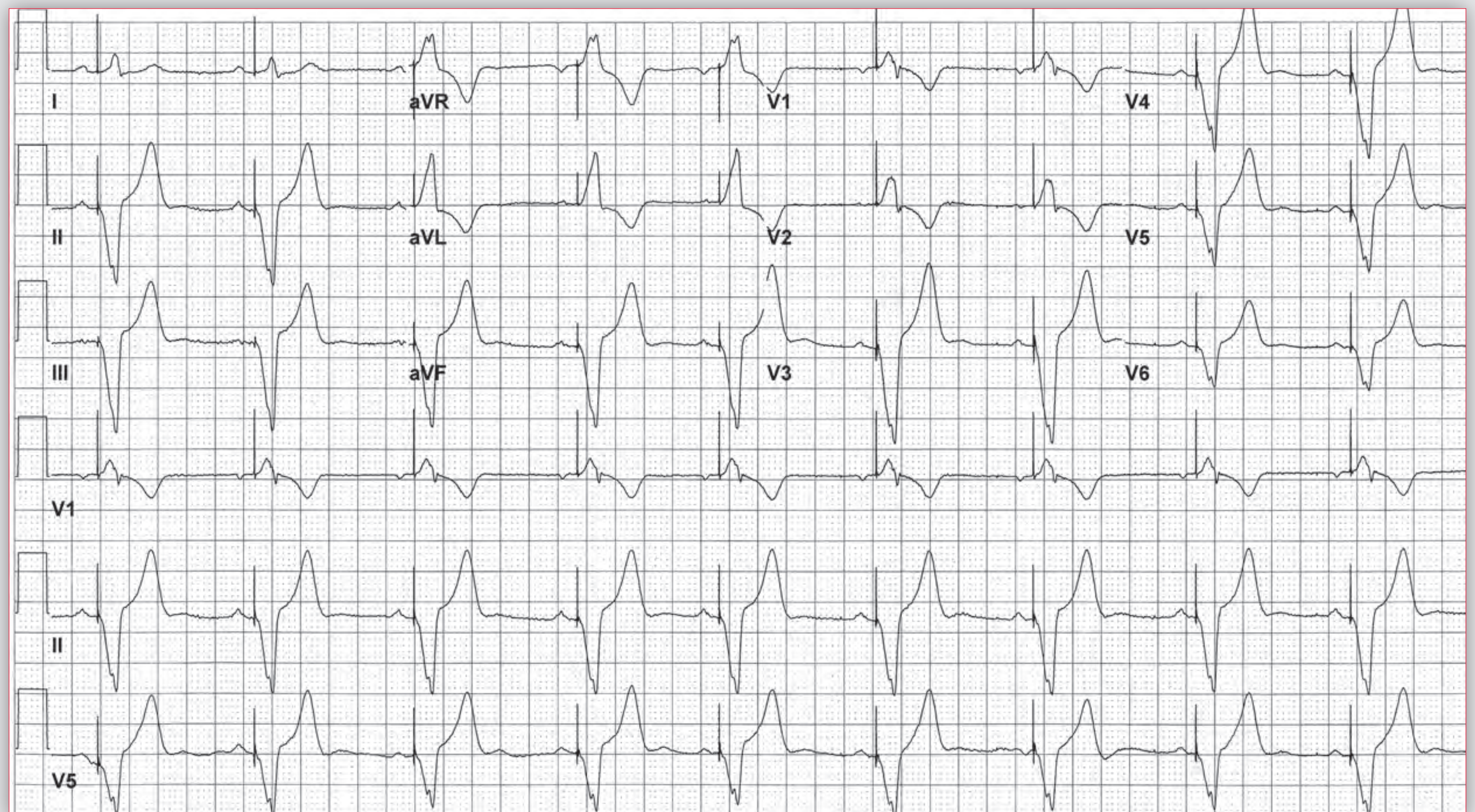
He is otherwise healthy without medical diagnoses. He does not take any medications.

As part of a comprehensive workup, his primary doctor obtains an ECG (ECG 42A).

Based on the findings, he is admitted to hospital.

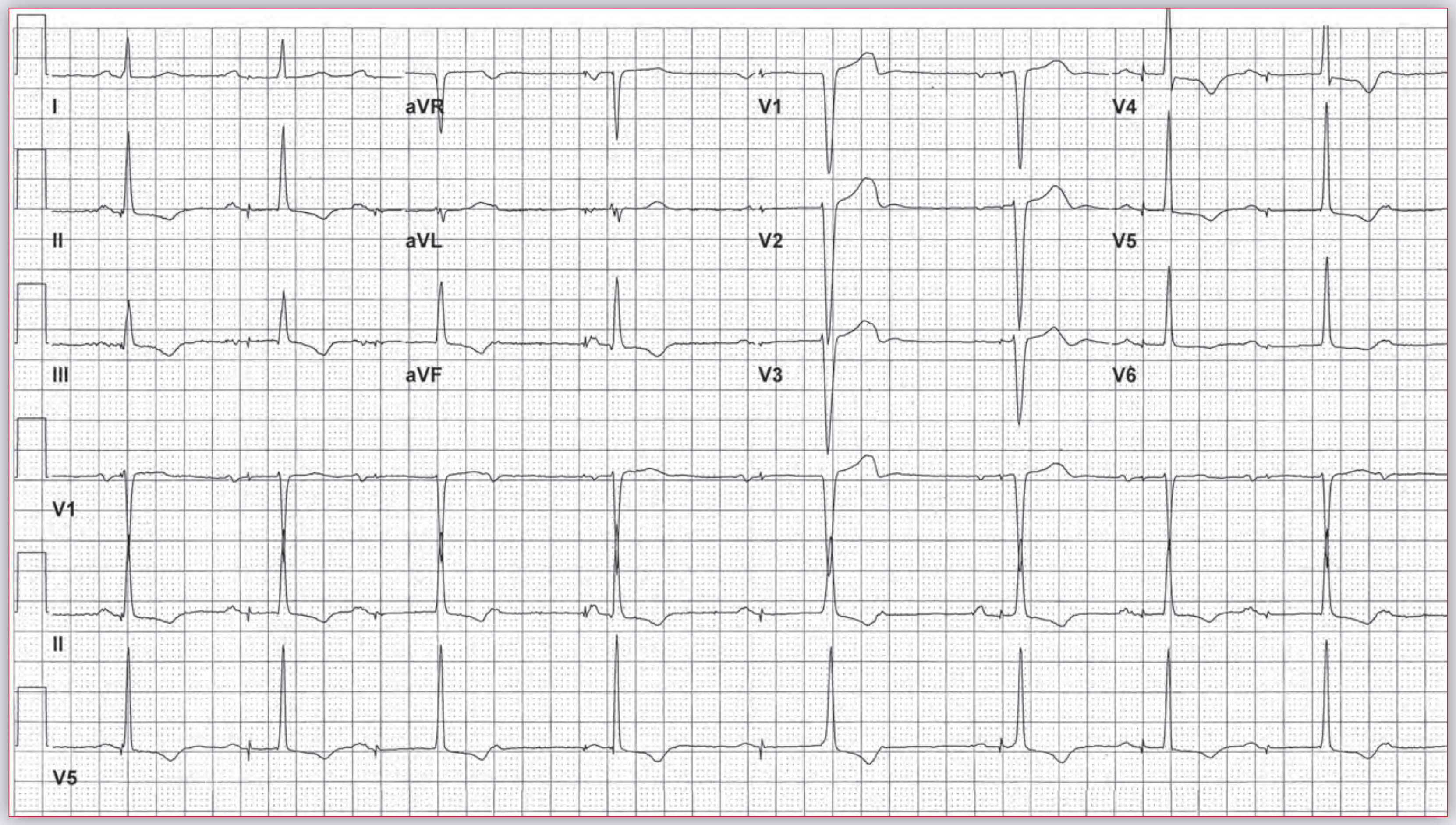
The patient is discharged from hospital; however, several days later re-presents with intermittent lightheadedness and palpitations. An ECG is obtained in the emergency department (ECG 42C).

ECG 42B



Core Case 42

ECG 42C

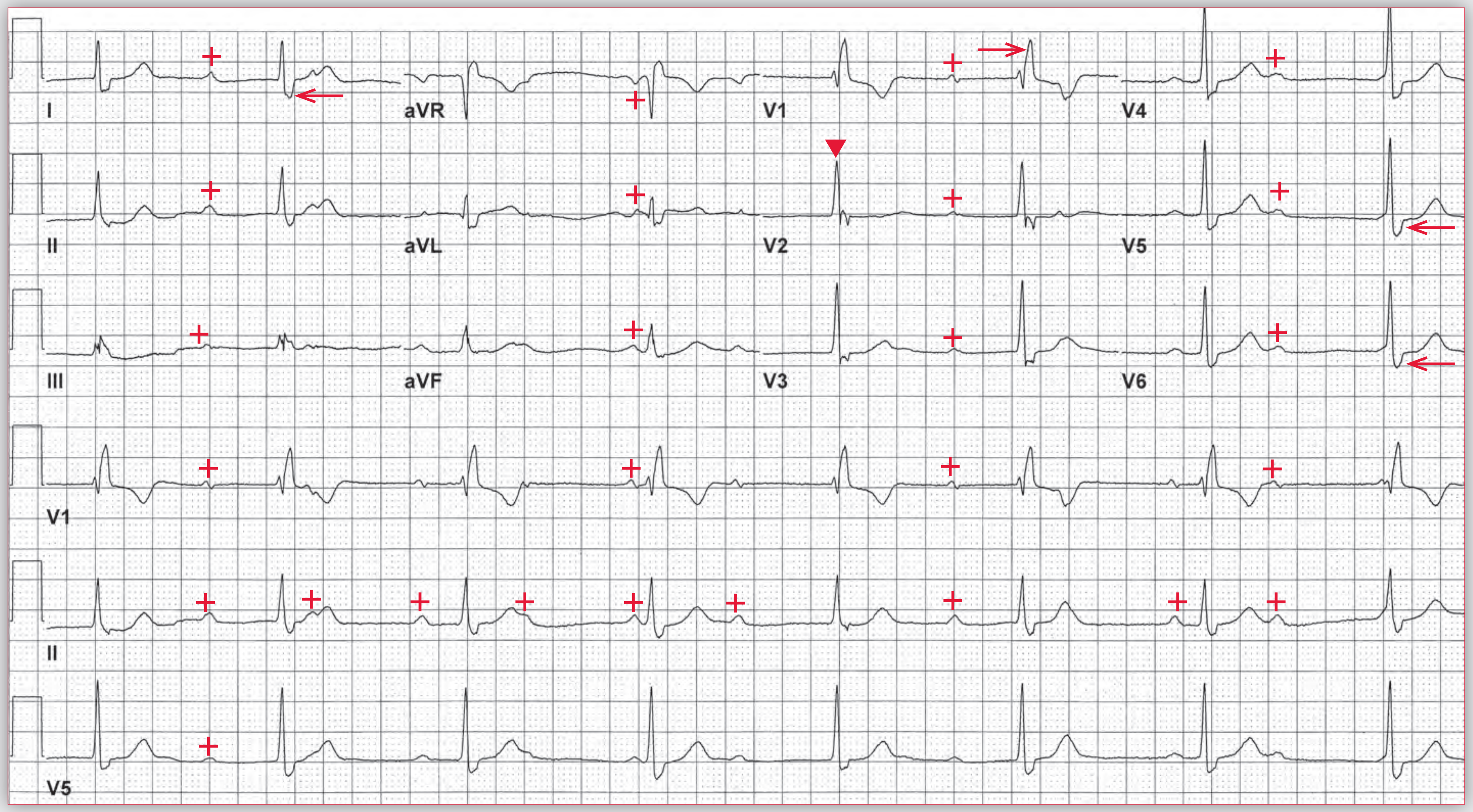


What abnormalities are noted, and what warranted hospital admission?

During admission, therapy is rendered, and a repeat tracing is obtained (ECG 42B). What new findings are noted?

Based on this history and ECGs, what is the patient's medical diagnosis?

What abnormalities are noted that would explain his symptoms?



ECG 42A Analysis: Sinus rhythm, AV dissociation, third-degree AV block, escape junctional rhythm with right bundle branch block, counterclockwise rotation

ECG 42A shows there is a regular rhythm with a rate of 48 bpm. There are P waves (+) seen, occurring at a rate of 86 bpm. The P waves are positive in leads I, II, aVF, V4–V6, and negative in aVR. Hence this is a sinus rhythm. The PR intervals are variable and hence there is AV dissociation present. As the atrial rate is faster than the ventricular rate, this is complete (third-degree) heart block. The QRS complex duration is wide (0.14 sec) and the morphology is that of a typical right bundle branch block (RBBB) with an RSR′ in V1 (→) and a broad S wave in leads I and V4–V6 (←). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (460/410 msec and 420/375 msec when the widened QRS complex duration is considered). There is early transition, with a tall R wave in lead V2 (▼). This is due to a shift in the electrical axis in the horizontal plane, determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces occur earlier in the precordium, accounting for early transition. Hence the escape rhythm is junctional with a right bundle branch block.

The development of complete heart block in a young person is very uncommon. Given the history of a fever, rash, arthralgia, and myalgia, the probable diagnosis is Lyme disease. Early Lyme disease is characterized by the appearance of the characteristic skin lesion, erythema migrans with or without constitutional symptoms. Symptoms include fatigue, malaise, lethargy, mild headache, mild neck stiffness, myalgias, arthralgias, and regional lymphadenopathy.

Weeks to months after infection, the patient may present with cardiac findings. Manifestations of cardiac involvement include AV nodal block, mild cardiomyopathy or myopericarditis. The most common abnormality is AV block, associated with Wenckebach or complete heart block. Very uncommon are other conduction abnormalities including fascicular or bundle branch blocks. Patients are may be symptomatic from the complete heart block, but the abnormality usually resolves after a few weeks. Temporary pacing may be required, but permanent pacemakers are not generally indicated.

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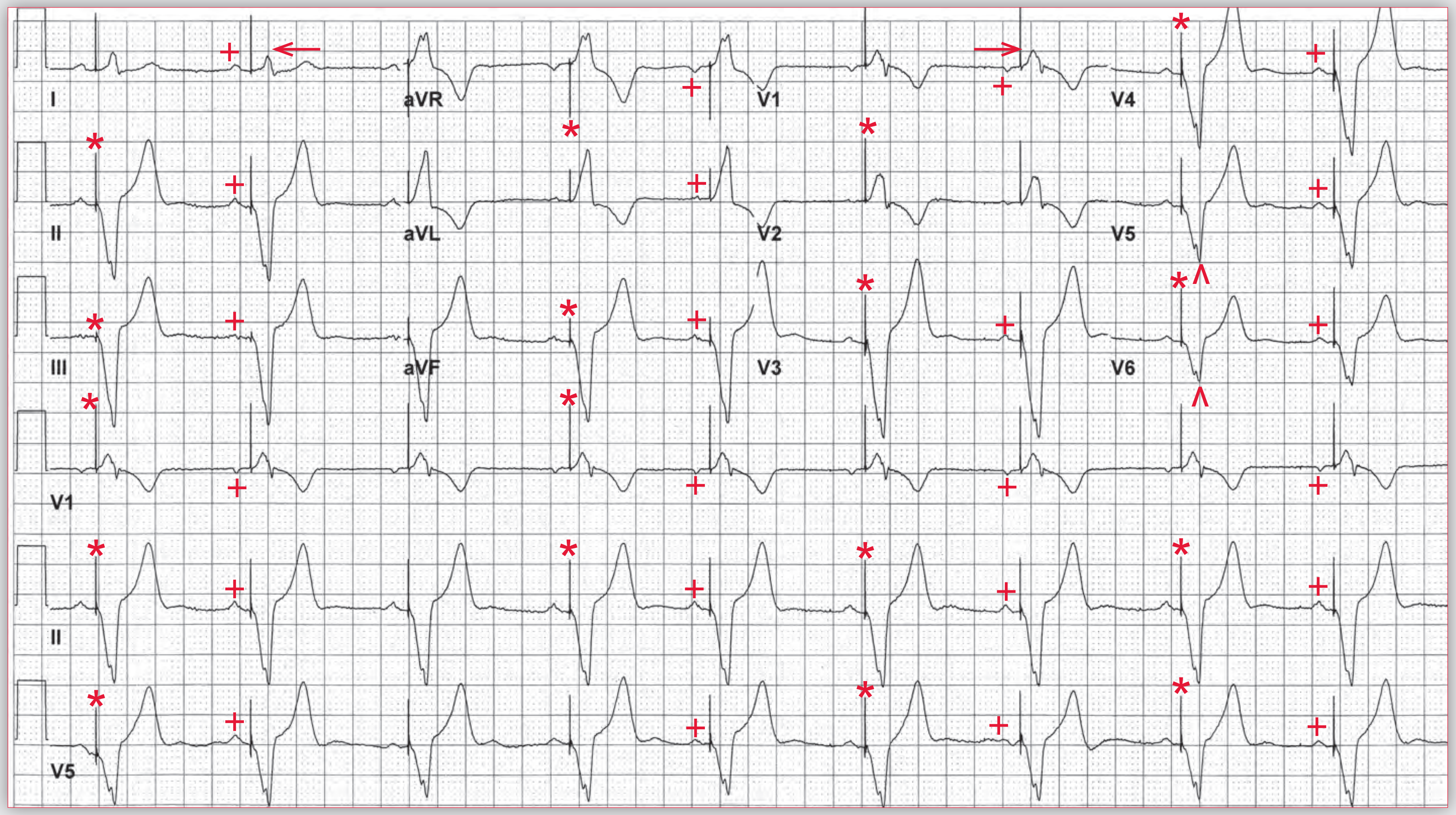

ECG 42B Analysis: Sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous and right ventricular pacing)

ECG 42B shows the diagnosis of Lyme disease was not entertained and as a result of symptomatic complete heart block the patient received a pacemaker, as indicated in ECG 42b. There is a regular rhythm with a rate of 62 bpm. Each QRS complex is preceded by a P wave (+) that is positive in leads I, II, aVF, and V4–V6 and negative in aVR. Hence there is a sinus rhythm present. There is a pacing stimulus (*) seen before each QRS complex and the "PR interval" (or AV delay) is stable (0.18 sec). The QRS complex duration is wide (0.16 sec) and the QT/QTc intervals are prolonged (480/490 msec) but are normal when the prolonged QRS complex duration is considered (420/425 msec). There is a broad R wave in lead I (←) indicating that the lead is in the right ventricle, even though there is a broad R wave in lead V1 (→) and a QS complex in leads V5–V6 (^). A tall R wave may be seen with a right ventricular lead that is located at or near the septum. Lead I is the most important lead to establish right versus left ventricular (biventricular pacing) as it is the only bipolar lead that looks at the impulse in a right to left direction. Therefore, any impulse originating in the right ventricle must have a positive QRS in lead I and an impulse originating in the left ventricle will have a negative QRS complex in this lead.

The pacemaker is dual chamber and is functioning in an atrial sensed, ventricular paced (P-wave synchronous or P wave activated) mode.

As indicated, AV conduction abnormalities with Lyme disease are most often transient and permanent pacing is not generally needed. Unfortunately the diagnosis of Lyme disease was not considered, and the permanent pacemaker was inserted because of symptomatic complete heart block, likely determined to be permanent.

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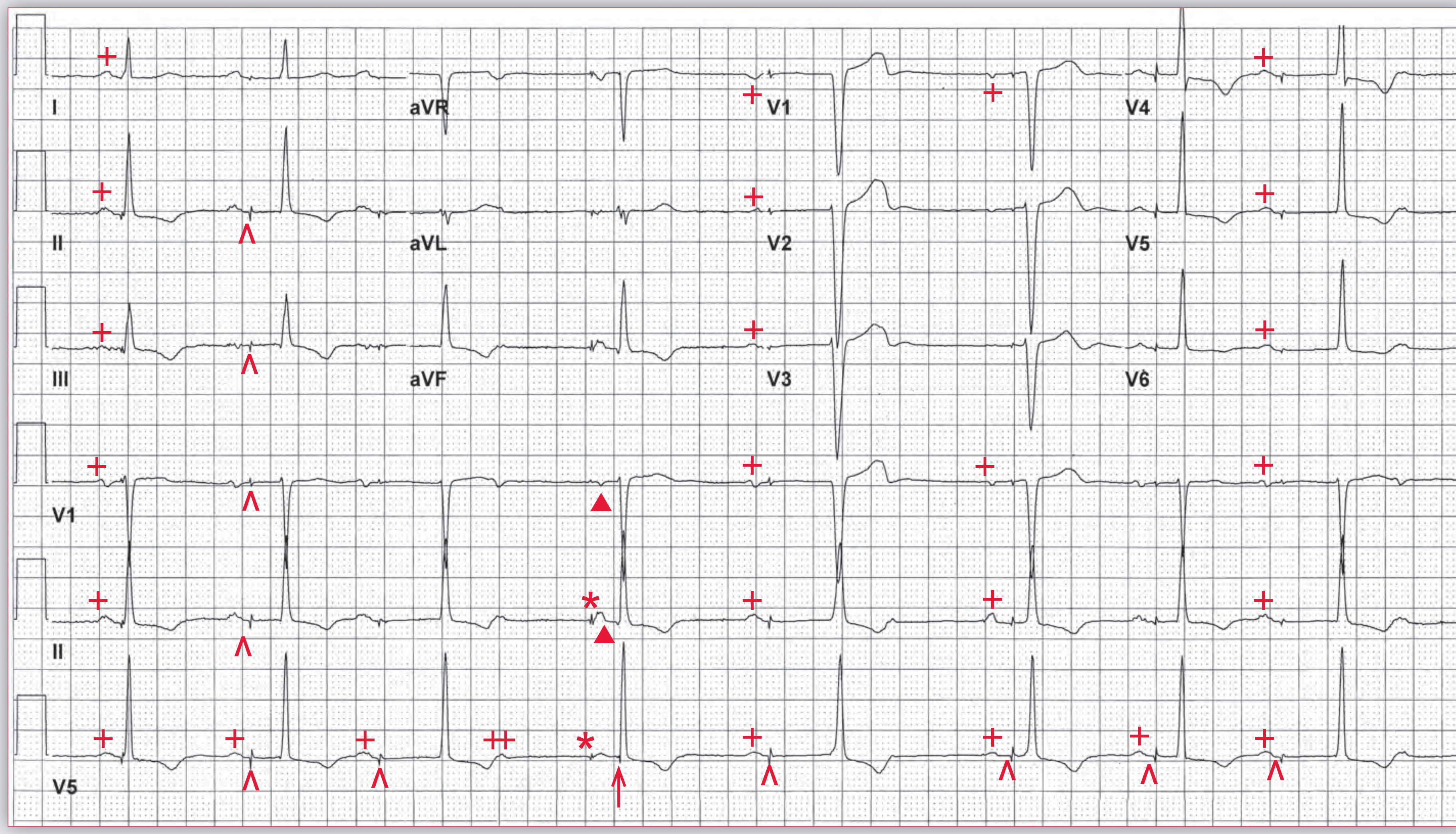


ECG 42C Analysis: Sinus rhythm with complete AV block with escape junctional rhythm and failure of ventricular capture

Two days after pacemaker insertion, the patient presented with a slow heart rate and the **ECG 42C** showed QRS complex with a normal duration (0.10 sec) at an average rate of 48 bpm. The QT/QTc intervals are normal (440/390 msec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). P waves (**+**) are present at a rate of 64 bpm and they are positive in leads I, II, aVF, and V4–V6, *ie*, a sinus rhythm. However, the PR interval is not constant and there does not appear to be any relationship between the P waves and QRS complexes, *ie*, there is AV dissociation present. As the atrial rate is faster than the ventricular rate, this is third-degree or complete heart block with an escape junctional rhythm. Pacing stimuli (^) at a rate of 64 bpm and the interval between the P wave and the pacing stimulus is constant (0.18 sec), which is identical to the PR interval (AV delay) seen in ECG 42B. Hence there is intact atrial sensing resulting in a ventricular stimulus (*ie*, atrial sensed, ventricular paced or P-wave synchronous ventricular pacing). However, there is no ventricular capture as no QRS complexes are seen after the pacemaker stimulus. Hence this is failure of the ventricular lead to capture.

After the third QRS complex, there is a slight pause. It can be seen that there is a P wave that is not sensed (**++**) as it is early after the QRS complex hence within the blanking period of the pacemaker (based upon the PVARP or post-ventricular atrial refractory period). As a result of the nonsensed P wave there is no ventricular pacemaker stimulus after this P wave. There is an atrial stimulus (*) and P wave (▲) before the fourth QRS complex. This P wave has a different morphology and is the result of the paced P wave. There is a ventricular stimulus (↑) that follows, after the appropriate AV delay (0.18 sec), indicating AV sequential pacing. However, this stimulus does not result in a paced QRS complex, further confirming failure to capture. Therefore, the noncaptured pacing stimuli are ventricular and there is failure of the ventricular lead to capture. The atrial lead appears to be sensing and capturing appropriately.

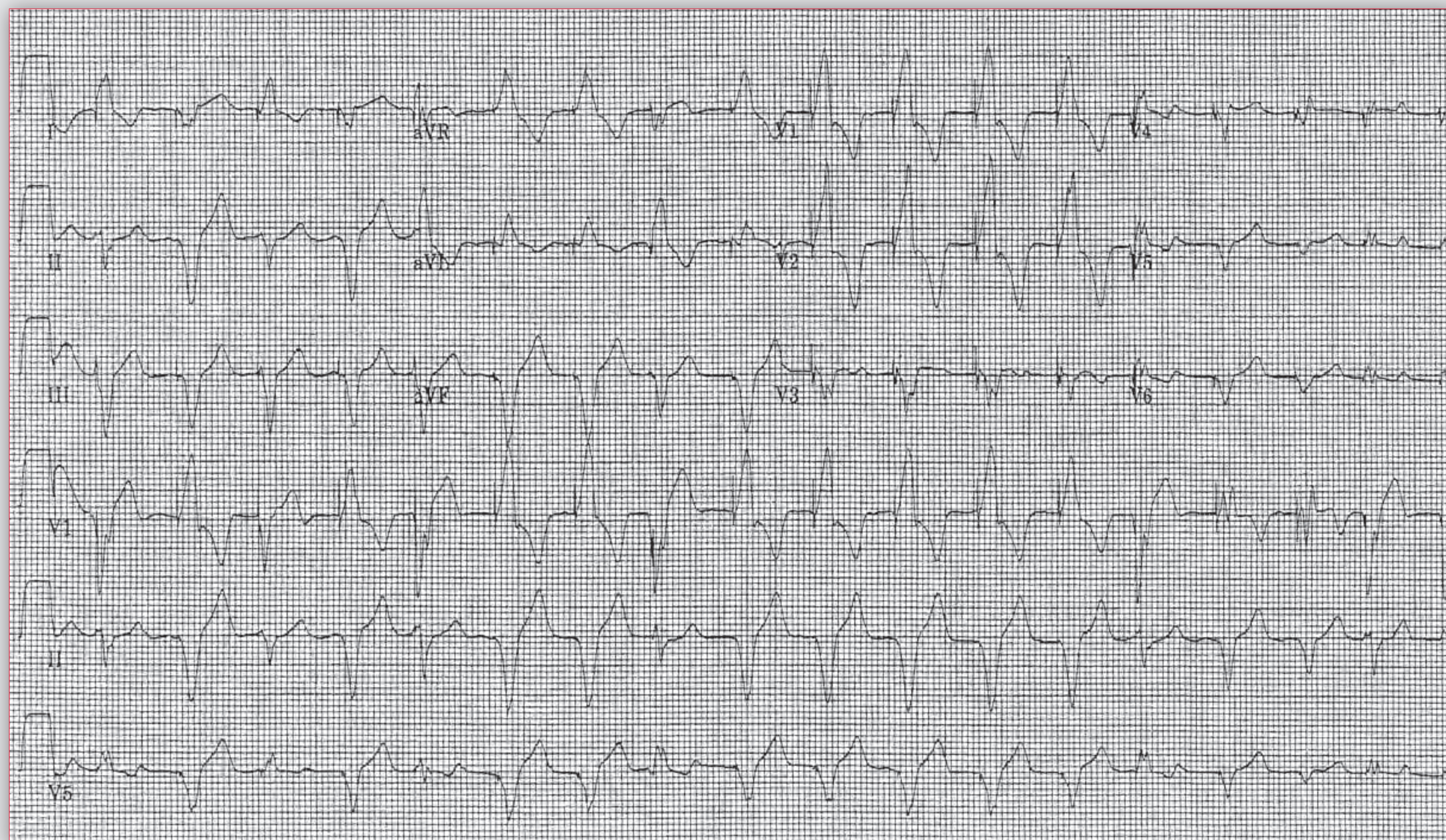
As the pacemaker was just recently implanted, it is most likely that the right ventricular pacing lead has lost adequate contact with the ventricular myocardium and needs to be repositioned. Another possibility is that as the result of inflammation and fibrosis at the tip of the pacing electrode the pacing threshold has increased, *ie*, a greater amount of energy output is necessary to capture the myocardium. Hence the pacing output can be increased.

What is unusual that on ECG 42A, the escape junctional rhythm had a RBBB morphology, while on ECG 42C, the escape junctional rhythm has a normal QRS complex duration. Although it is possible that this represents a rate-related RBBB, the rates of the junctional rhythms are similar. Another possibility is that the RBBB is also a result of Lyme disease, although as mentioned above this is uncommon. ■

Core Case 43

A 64-year-old man with a dilated cardiomyopathy and a left ventricular ejection fraction (LVEF) of 25% had a biventricular implantable cardioverter-defibrillator (ICD) inserted one year ago, He was last seen by his cardiologist 1 month ago, and at the time he was clinically stable. His ECG showed a regular rate of 60 bpm with paced complexes that were consisted with a biventricular pacemaker. He presents to the emergency department with a history of shortness of breath and fatigue. These symptoms have been present for 2 weeks and have become progressively worse. He also notes weight

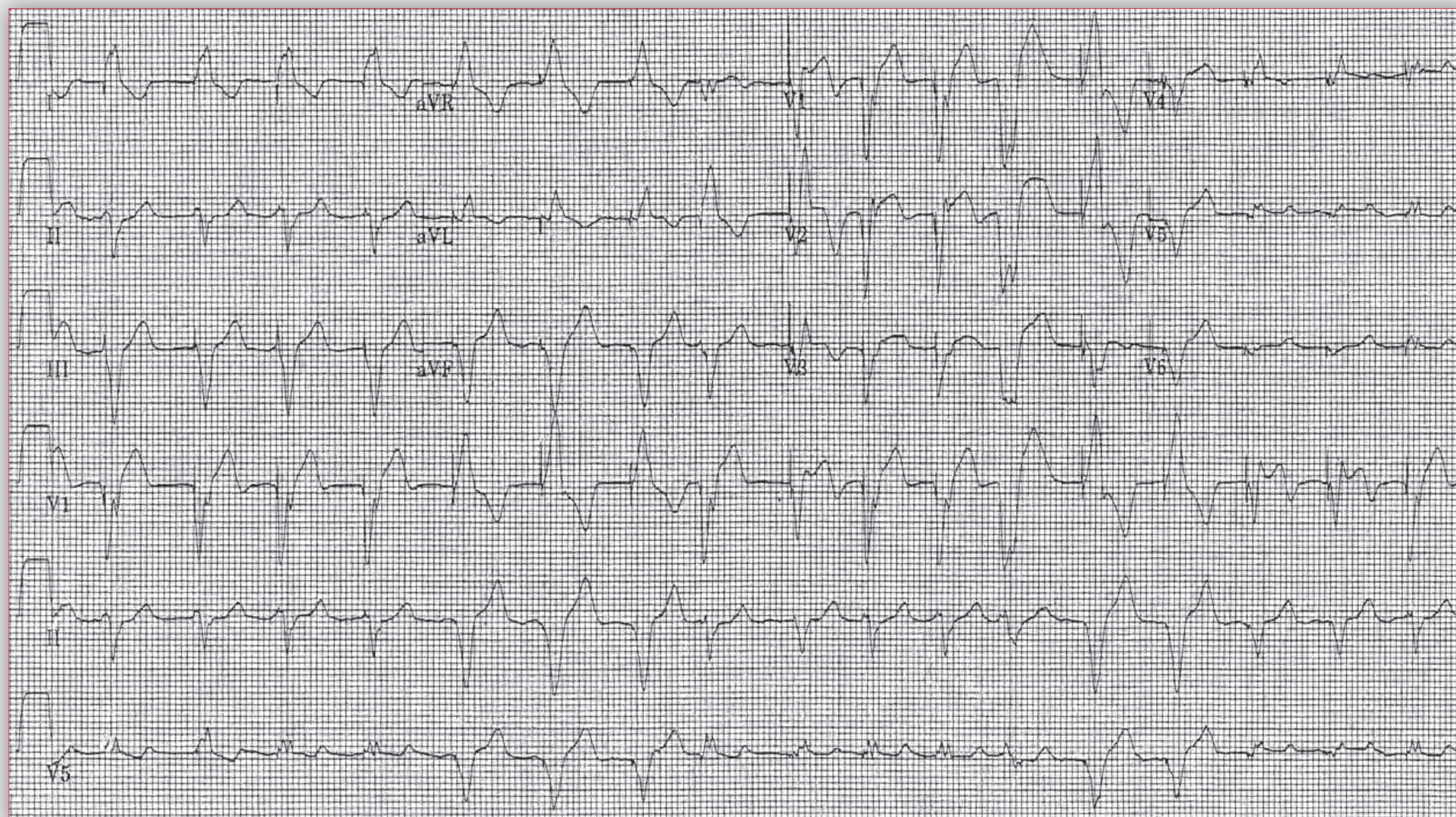
ECG 43A



gain of 8 pounds over these 2 weeks. Physical examination finds bilateral rales, 2+ bilateral peripheral edema, neck vein distention and a heart rate of 120 bpm. A chest x-ray demonstrates pulmonary edema. The admission ECG (ECG 43A) is shown. He is treated with intravenous furosemide and puts out 2 liters. On the following day, he states that his breathing is improved, and he is symptomatically better. Another ECG (43B) is obtained because of a persistent tachycardia.

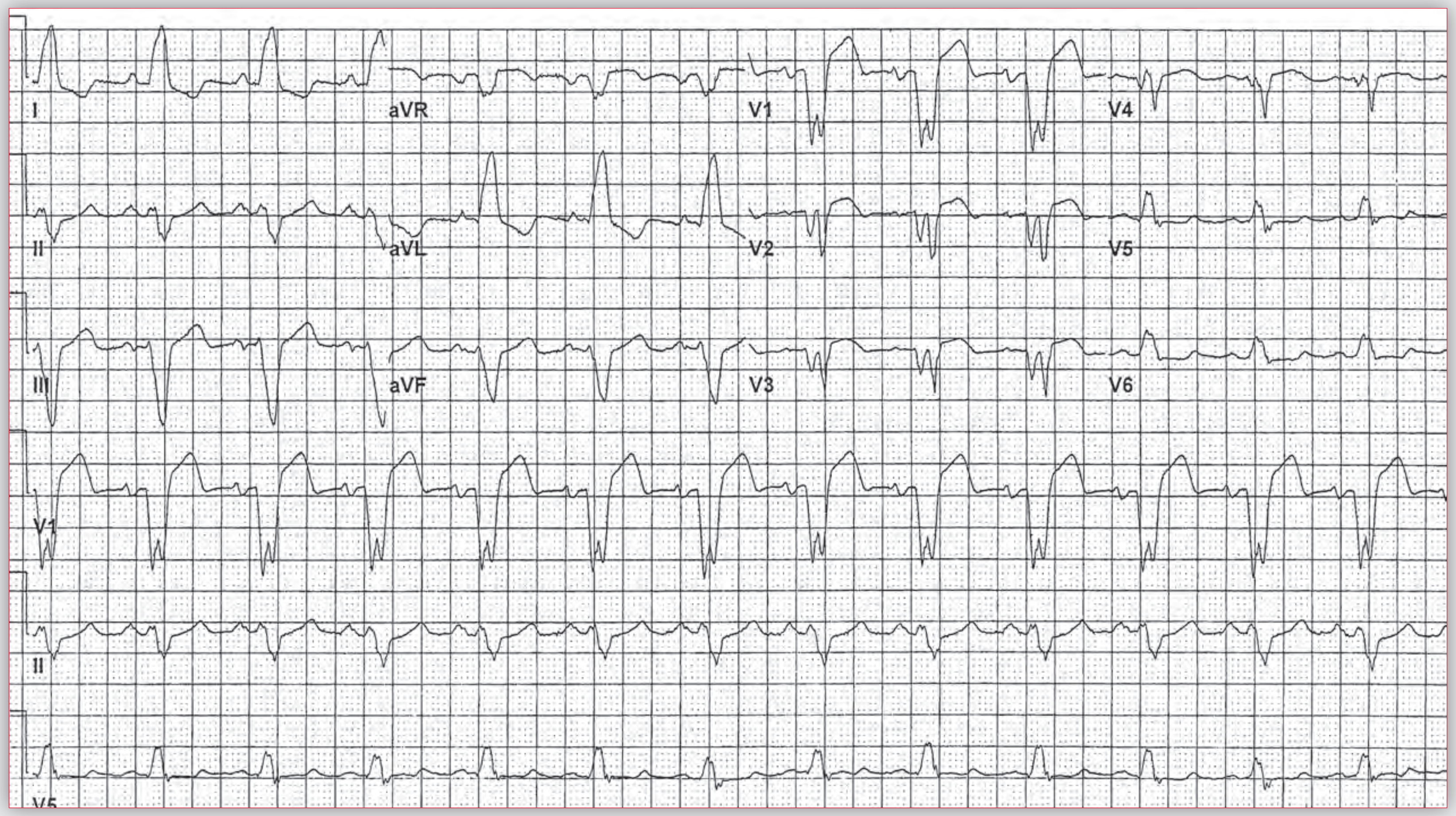
The patient's ECG prior to insertion of the ICD is also shown (ECG 43C).

ECG 43B



Core Case 43

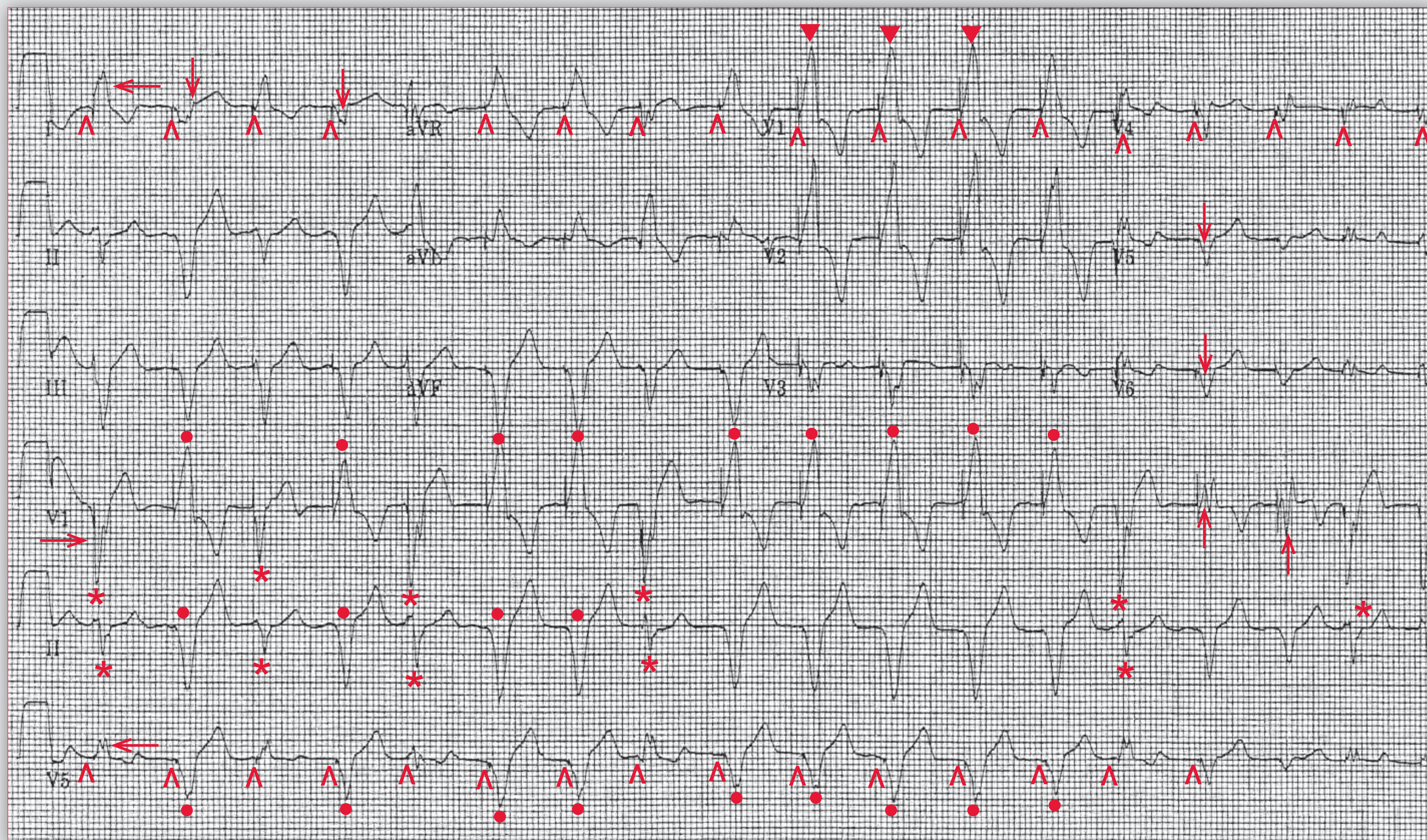
ECG 43C



Core Case 43

Is the pacemaker function normal?

If not, what is the abnormality seen?



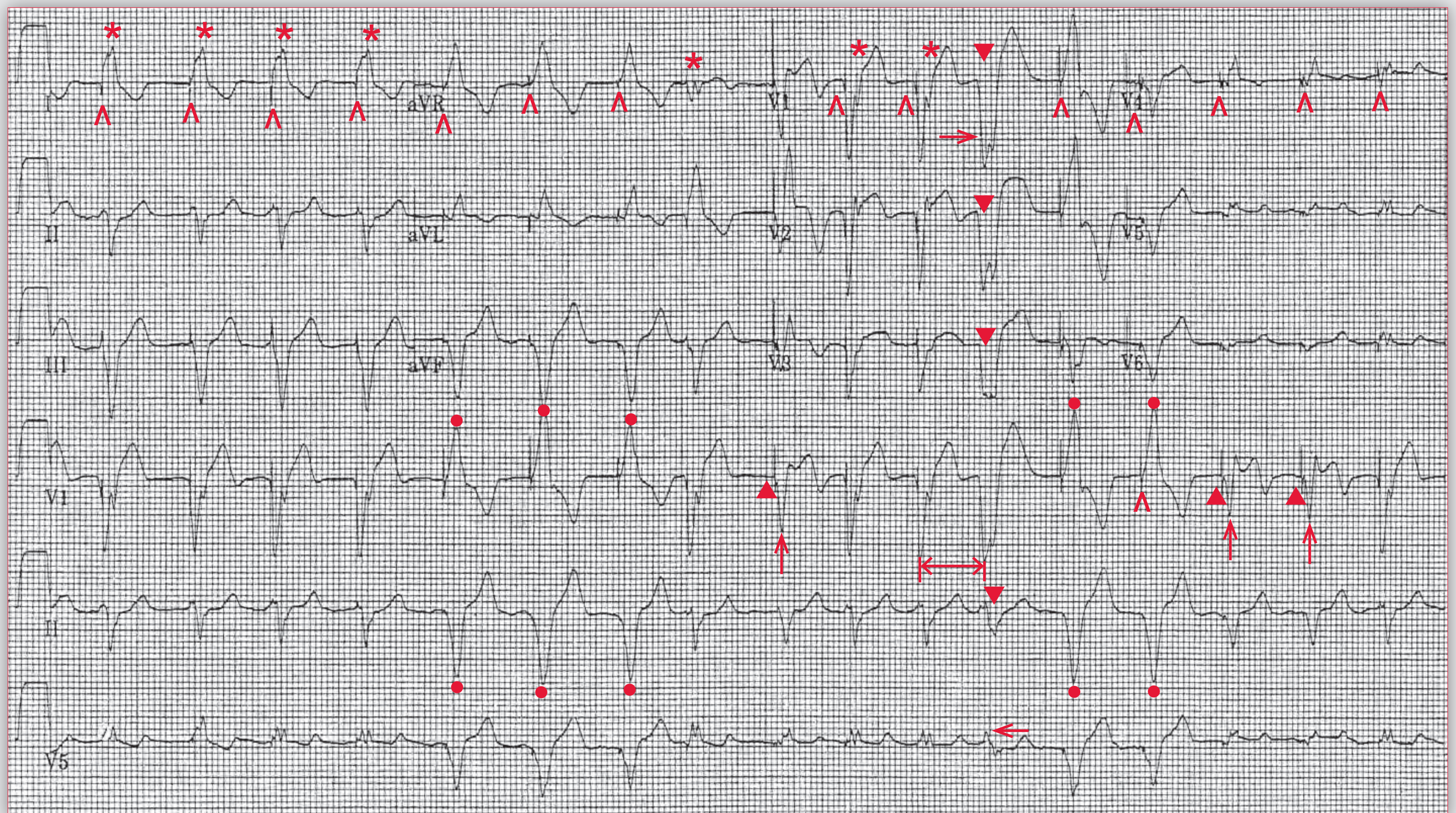
ECG 43A Analysis: Atrial fibrillation, biventricular pacemaker, atrial sensed, ventricular paced, intermittent failure of left ventricular lead capture (left and right ventricular pacing), pseudofusion

ECG 43A shows there is a regular rhythm with a rate of 110 bpm. No atrial activity is seen. There is a pacemaker stimulus seen before each of the QRS complexes (^), The QRS complexes have two different morphologies. Complexes 1, 3, 5, 8, 14, and 17 (*) are wide (0.14 sec) and have a left bundle branch block (LBBB) morphology with a broad R wave in leads I and V5 (←) and a QS complex in lead V1 (→). These QRS complexes have a morphology that is typical for right ventricular (RV) pacing. The remaining complexes (2, 4, 6, 7, and 9–13) (•) are also wide (0.16 sec) and have a QS complex in leads I and V5–V6 (↓) and a tall R wave in lead V1 (▼). Lead I is a bipolar lead that looks at the impulse as it travels from the right to the left arm. An impulse that goes from right to left generates a positive QRS complex (tall R wave) in lead I. This is seen with RV pacing. An impulse that goes from left to right is associated with an initial Q wave or a QS complex (negative QRS complex) in lead I. This is seen with left ventricular (LV) or biventricular pacing. In addition, LV pacing is associated with a tall R wave in lead V1 as the impulse is directed toward the right. Complexes 2, 4, 6, 7, 9–13 (•), which have this morphology, are thus the result of LV (biventricular) pacing. Therefore, there is intermittent failure of LV lead capture. When this occurs, there is only RV pacing. The QT/QTc intervals are prolonged (360/490 msec), but are normal when the prolonged QRS complex duration is considered (300/405 msec).

Since the pacing rate is 110 bpm, the ventricular stimuli are the result of tracking an atrial rhythm. As there is no evidence of atrial activity, the underlying rhythm is probably atrial fibrillation, and the pacemaker is tracking this arrhythmia at its upper rate limit. The fifteenth and sixteenth QRS complexes (↑) have a different morphology from the other QRS complexes, although they resemble the LV or biventricular paced complexes. It is possible that these complexes are fusion between the pacemaker and native conduction. This would also support the fact that the underlying rhythm is atrial fibrillation, with an occasional ventricular response rate that is slightly faster than 110 bpm, resulting in a native complex resulting from AV node–His-Purkinje conduction that fuses with a pacemaker stimulus.

It is likely that the patient has developed worsening heart failure as the result of atrial fibrillation and a persistent heart rate of 110 bpm due to continuous ventricular pacing. In addition, heart failure may have been exacerbated as a result of intermittent lack of capture of the left ventricular lead and the absence of biventricular pacing.

continues



ECG 43B Analysis: Atrial fibrillation, biventricular pacemaker, atrial sensed, ventricular paced, intermittent failure of LV lead capture (left and right ventricular pacing)

ECG 43B was obtained on the day after admission. The rate is slightly irregular. Although the rate is primarily 110 bpm, there are occasional shorter RR intervals at rates of 120 bpm. There is a pacemaker stimulus before each QRS complex (^), except for the twelfth QRS complex (▼). Similar to ECG 43A, some of the QRS complexes show right ventricular pacing (*) and other show LV or biventricular pacing (●). Therefore, intermittent lack of LV capture is still present. In addition, the rates of 100–120 bpm with the absence of atrial activity means that atrial fibrillation is still present.

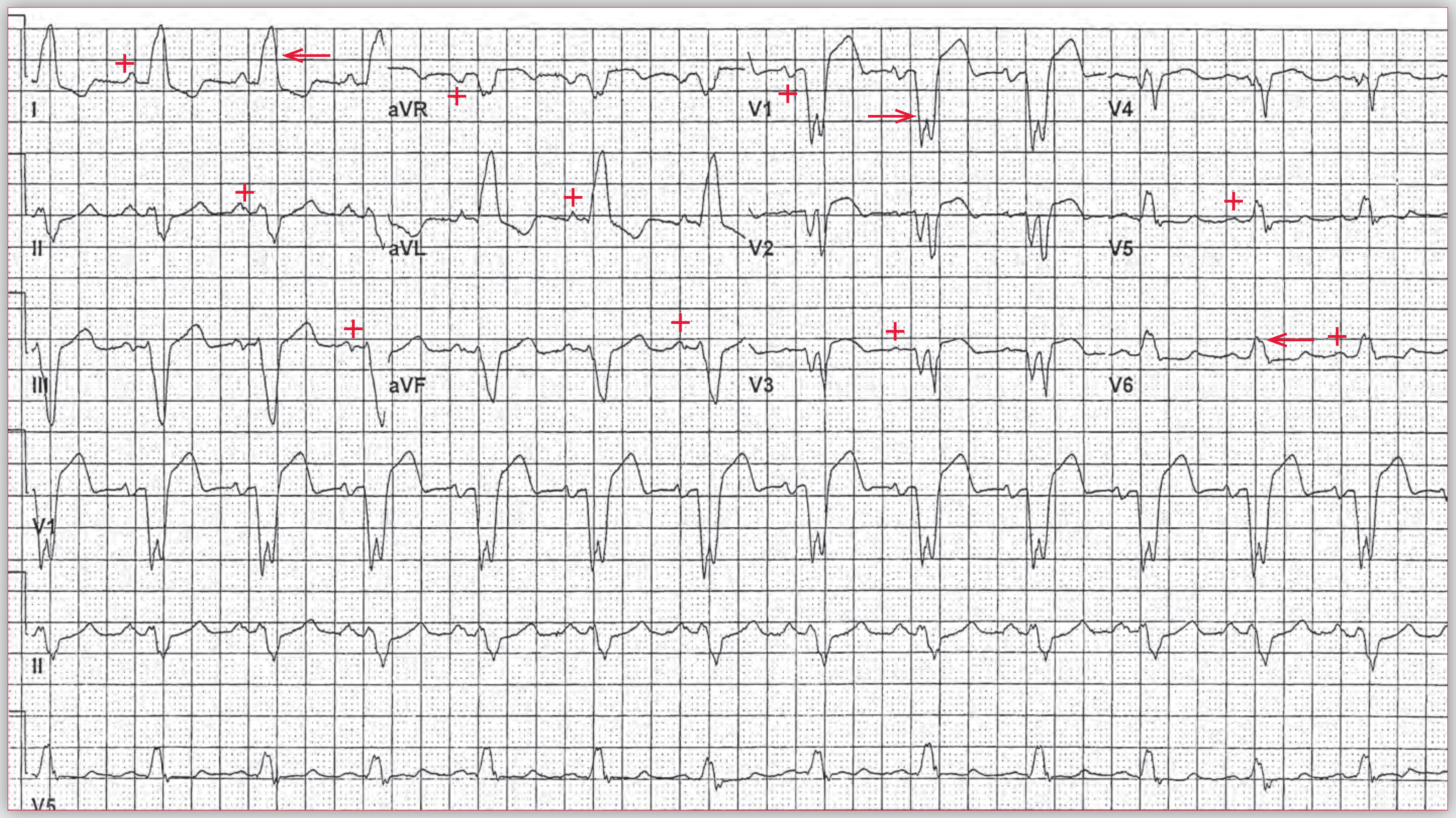
Of interest is the twelfth QRS complex (▼) that does not have a preceding ventricular pacemaker stimulus. This QRS complex occurs at a rate of 150 bpm (↔), and it has a morphology that is different than both the RV and LV paced QRS complexes, although the morphology is typical for a LBBB with a deep QS complex in lead V1 (→) and a broad R wave in lead V5 (←). In addition, it is wider (0.18 sec) than the paced QRS complexes and has a morphology of a typical LBBB. The absence of a pacemaker stimulus before this complex suggests that this is a native QRS complex that occurs at a rate faster than the upper rate limit of the pacemaker.

In addition, QRS complexes 9, 15, and 16 (↑), which are preceded by pacing stimuli (▲), also have a different morphology, best seen in the lead V1 rhythm strip. They are unlike the RV or LV paced complexes or the native QRS complex. This suggests that they are fusion complexes, *ie*, fusion between the native complex and a RV paced complex. This results from activation going through the AV node that fuses with the impulse generated by the RV pacing lead. This can occur if the ventricular rate of the atrial fibrillation is equal to the upper rate limit of the pacemaker.

Treatment of heart failure in this patient may be difficult in the presence of a continuous tachycardia with a rate of > 110 bpm. This is the result of the pacemaker tracking atrial fibrillation. The use of a magnet, which inactivates pacemaker-sensing capabilities, will change the pacing mode to DOO, or a fixed-rate pacemaker. There will be regular atrial and ventricular pacing stimuli occurring at the lower rate limit of the pacemaker. However, the pacemaker will no longer sense atrial fibrillation and pace the ventricles at a rapid rate, and the ventricular rate will be based entirely upon AV nodal conduction. Rate control can be achieved with an AV nodal blocking agent. In addition, the LV lead needs to be revised.

Most pacemakers have a programmable feature known as mode switching. When a rapid atrial rate is sense, the pacemaker automatically changes to a VVI mode, or demand ventricular pacing. This will terminate tracking of the atrial arrhythmia and the ventricular response rate to the atrial fibrillation will be based entirely on AV nodal conduction.

continues



ECG 43C Analysis: Normal sinus rhythm, LBBB

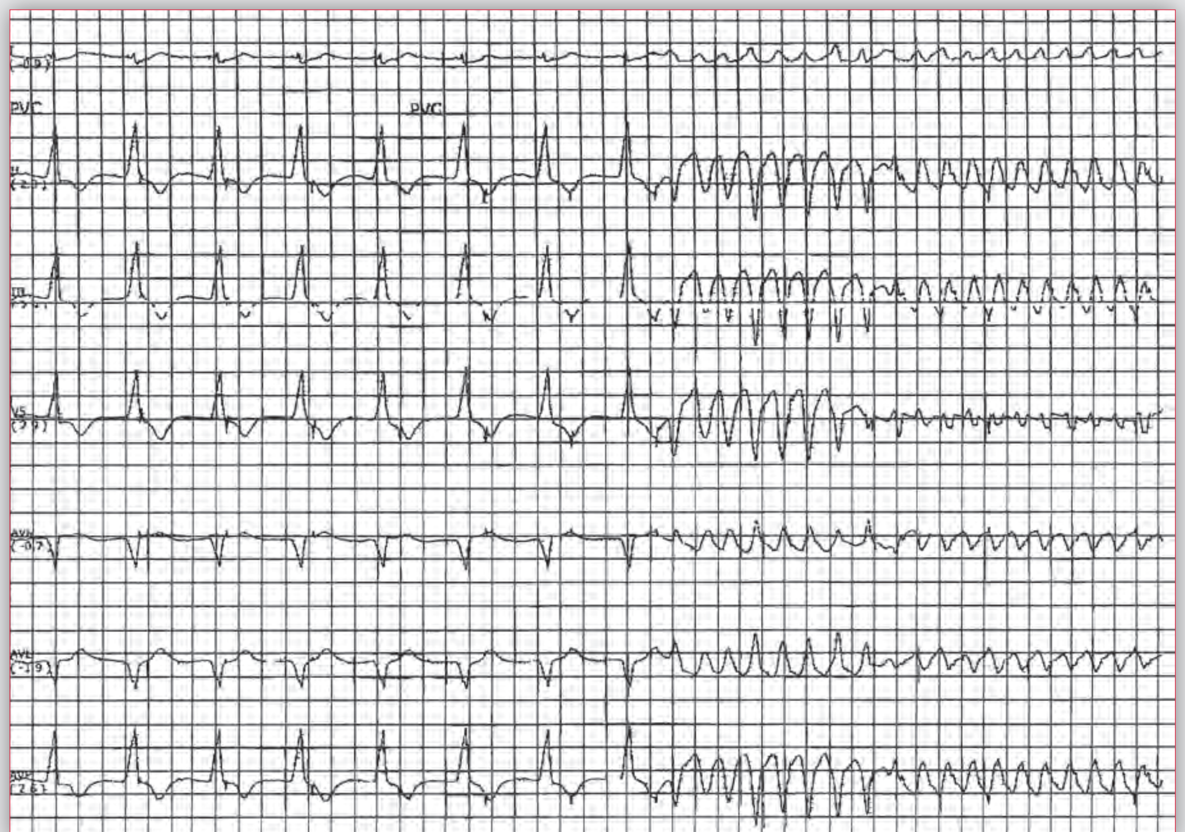
ECG 43C is from the same patient as ECGs 43A and 43B and is the baseline ECG prior to the insertion of the biventricular ICD. There is a regular rhythm at a rate of 76 bpm. There is a P wave before each QRS complex (**+**) with a stable PR interval of 0.18 sec. The P waves are positive in leads I, II, aVF, and V5–V6. This is a normal sinus rhythm. The QRS complex duration is increased (0.18 sec), and the morphology is typical for a LBBB with a broad R wave in leads I and V6 (←) and a QS complex in lead V1 (→). The QT/QTc intervals are prolonged (440/495 msec) but are normal when the prolonged QRS complex duration is considered (360/405 msec). The QRS complex morphology is identical to that of the twelfth QRS complex in ECG 43C, confirming that this complex was indeed a native complex.

It is common for the QRS complex generated by biventricular pacing to have a duration that is less than the duration of the native QRS complex. This is because of simultaneous (or almost simultaneous) activation of left and right ventricles, which reduces the degree of intraventricular conduction delay. ■

Notes

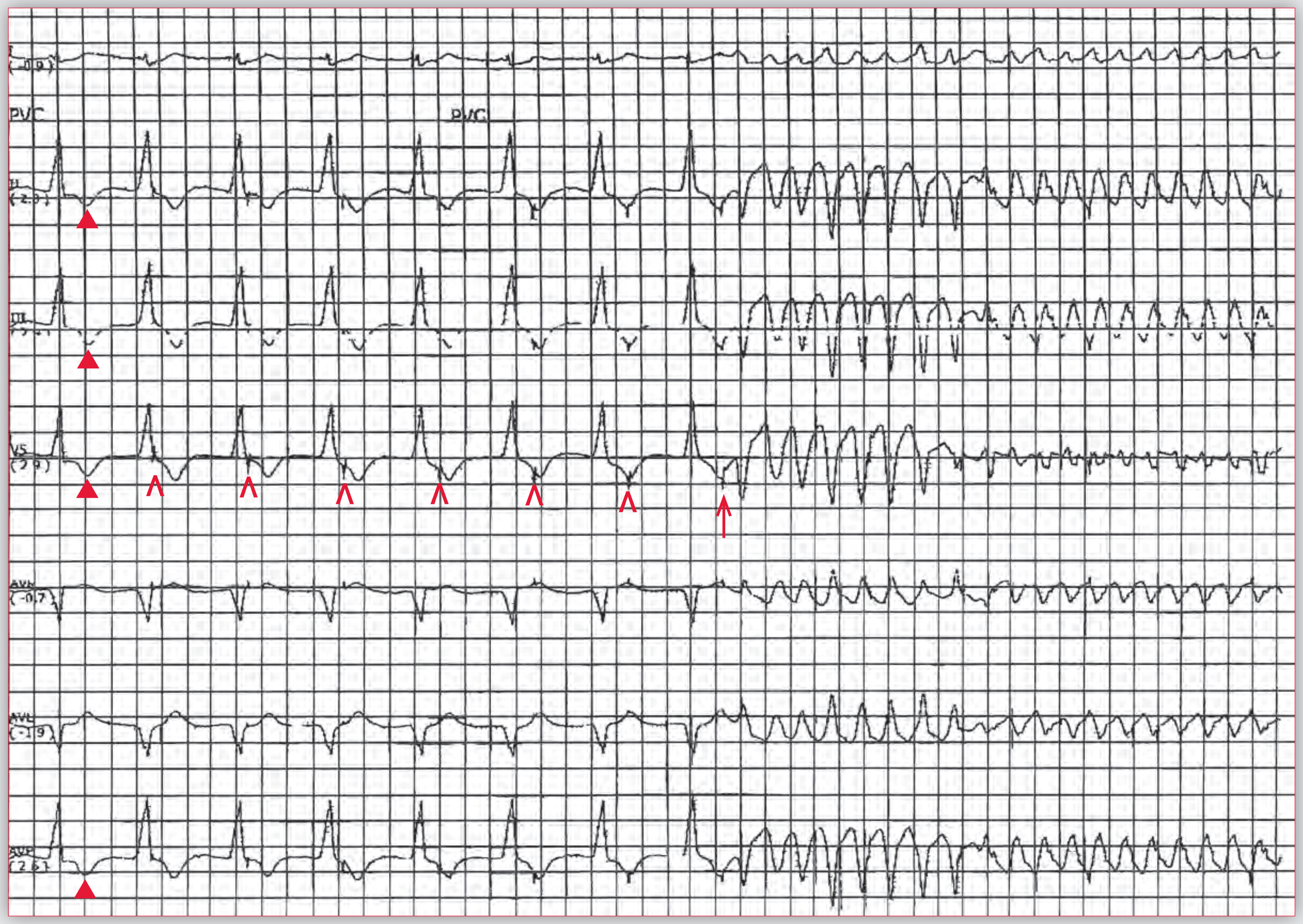
Core Case 44

A 72-year-old man is admitted to the hospital for an elective carotid endarterectomy. He has a history of coronary artery disease and had two stents placed into the right coronary artery 2 years ago. He has been stable since that time. He has a single-chamber ventricular pacemaker that was placed for a sick sinus syndrome. Prior to surgery the pacemaker is reprogrammed to a VOO mode to avoid sensing of electrical stimuli during the surgical procedure. The surgery is uncomplicated. While in the recovery room, he suddenly develops severe substernal chest discomfort, and an ECG shows new symmetric T-wave inversions in leads II, III, aVF, and V5–V6. He received sublingual nitroglycerin and intravenous β-blocker. Although the pain decreases, he still has residual chest discomfort. Cardiology is consulted and there is a discussion about urgent cardiac catheterization. Several minutes later, there is a change in his rhythm noted on telemetry, and then he suddenly loses consciousness. CPR is begun and is successful. He is brought to the catheterization laboratory, and angiography demonstrates an acute thrombus in the mid right coronary artery, distal to the previous stent.



What does the telemetry strip show?

What is the etiology for the arrhythmia?



ECG 44 Analysis: **Junctional rhythm, ventricular pacemaker with failure to sense (VOO mode), "R-on-T" pacemaker stimulus, polymorphic ventricular tachycardia**

Initially, there is a regular rhythm at a rate of 84 bpm. There is no evidence of atrial activity. The QRS complexes have a normal duration (0.10 sec), and the axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal and the QT/QTc intervals are normal (360/425 msec). There are T-wave inversions (▲) in leads II, III, aVF, and V5. Pacemaker stimuli are seen (^) at a rate of 76 bpm, but they have a variable relationship to the QRS complexes. The pacemaker stimulus following the eighth QRS complex is seen just after the apex of the T wave (↑). Immediately following this, there is a rapid polymorphic ventricular tachycardia that manifests changes in QRS morphology and axis.

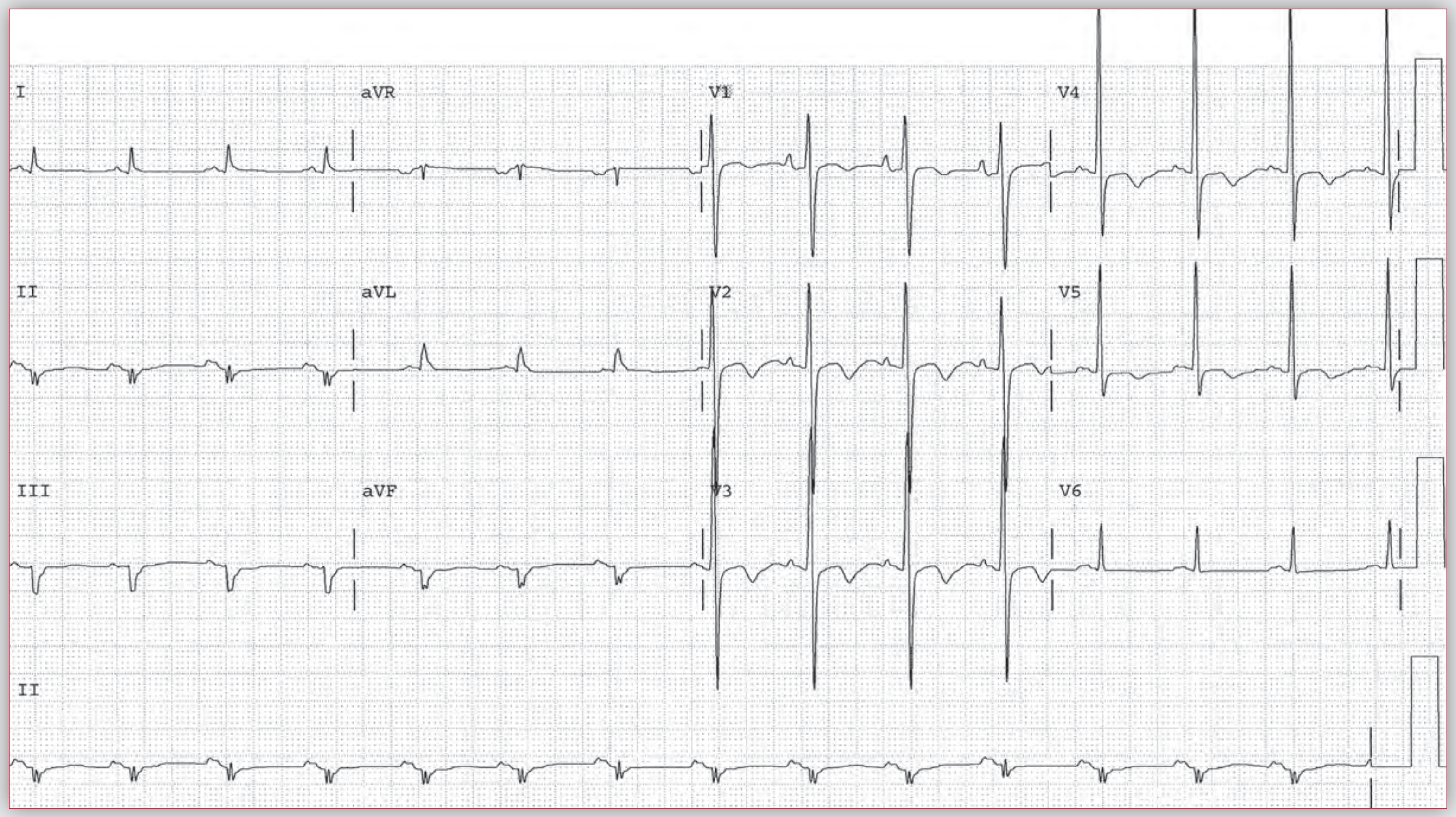
The pacemaker was still in a VOO mode after surgery, resulting in fixed-rate ventricular pacing at a rate of 76 bpm. As ventricular sensing has been inactivated, the ventricular lead does not sense the native QRS complexes that occur at a faster rate (84 bpm). Hence the pacemaker stimuli occur at variable times in relation to the native QRS complexes. It can be seen that they are occurring on the T wave, and after the eighth QRS complex, the pacemaker stimulus (↑) occurs right after the apex of the T wave. This is known as the "R-on-T" phenomenon, and it correlates with the vulnerable period of the action potential, which is the end of phase 3 and the very beginning of phase 4. During this time, the action potential transiently becomes more negative that –90 mV, or is hyperpolarized. It is during this time when the membrane is vulnerable to electric stimuli and when ventricular fibrillation is more easily induced. The amount of energy necessary to induce this arrhythmia is termed the VF threshold. Under normal situations, a large electrical current is necessary to induce ventricular fibrillation (or polymorphic ventricular tachycardia), *ie*, the ventricular fibrillation threshold is high. However, the threshold is lowered in the presence of ischemia, and a small current strength can induce ventricular fibrillation. In a nonischemic heart, the output of a pacemaker is not adequate to provoke ventricular fibrillation. However, in the presence of ischemia, the output from a pacemaker can induce this arrhythmia. The pacemaker should be promptly changed to a VVI mode to avoid the stimulus from falling on the T wave. Appropriate therapy for an acute coronary syndrome should also be initiated. ■

Core Case 45

A 36-year-old male with an unknown past medical history is admitted to the intensive care unit in cardiogenic shock. Echocardiography documents markedly reduced left ventricular ejection fraction (LVEF). An ECG is read by the house officer (ECG 45A) and possible etiologies for the patient's condition are suggested, including a hypertrophic cardiomyopathy.

The attending physician glances at the ECG, obtains a repeat study (ECG 45B), and suggests a completely different set of etiologic possibilities, namely inflammatory, infiltrative, or dilated cardiomyopathy.

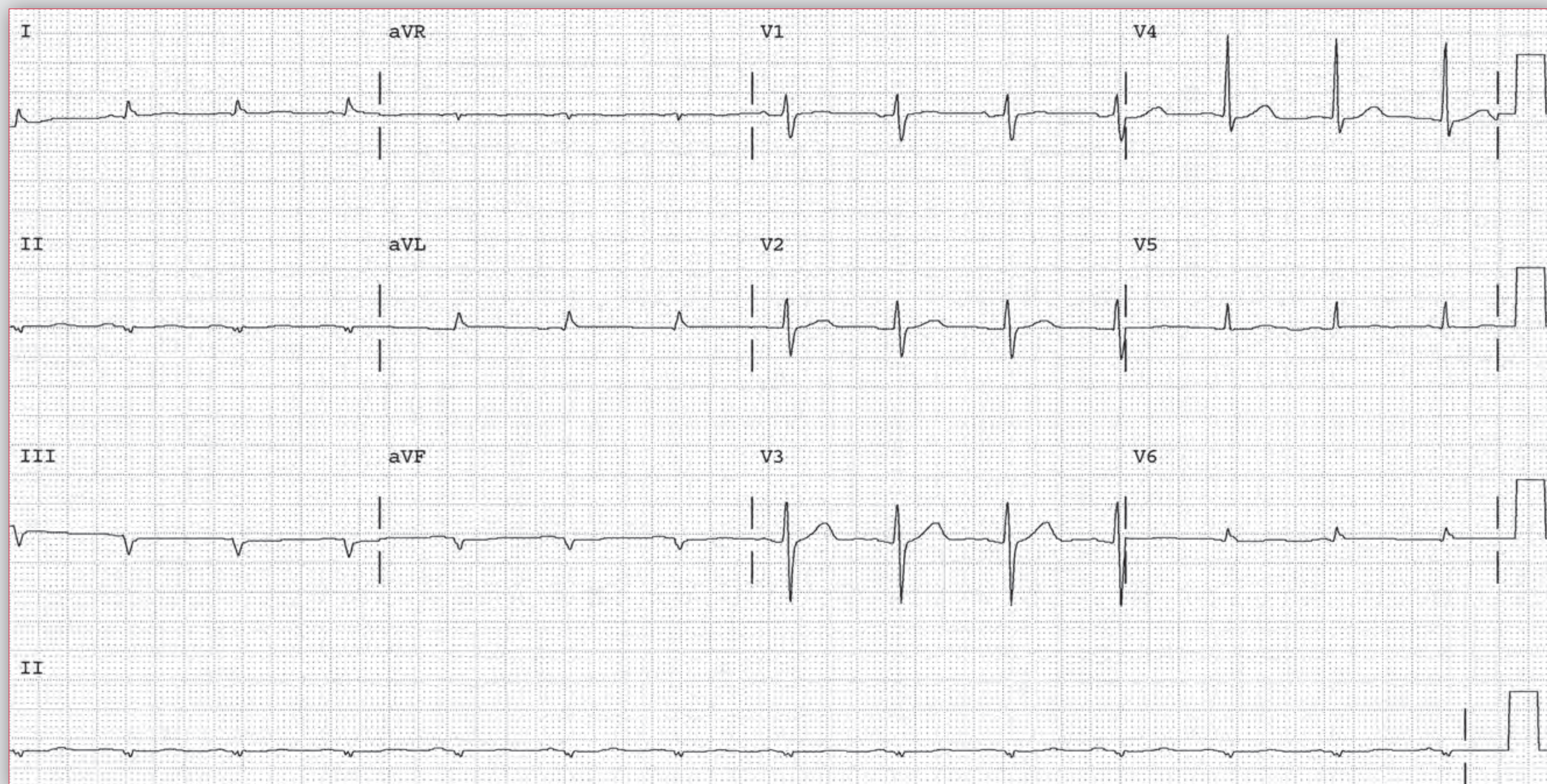
ECG 45A

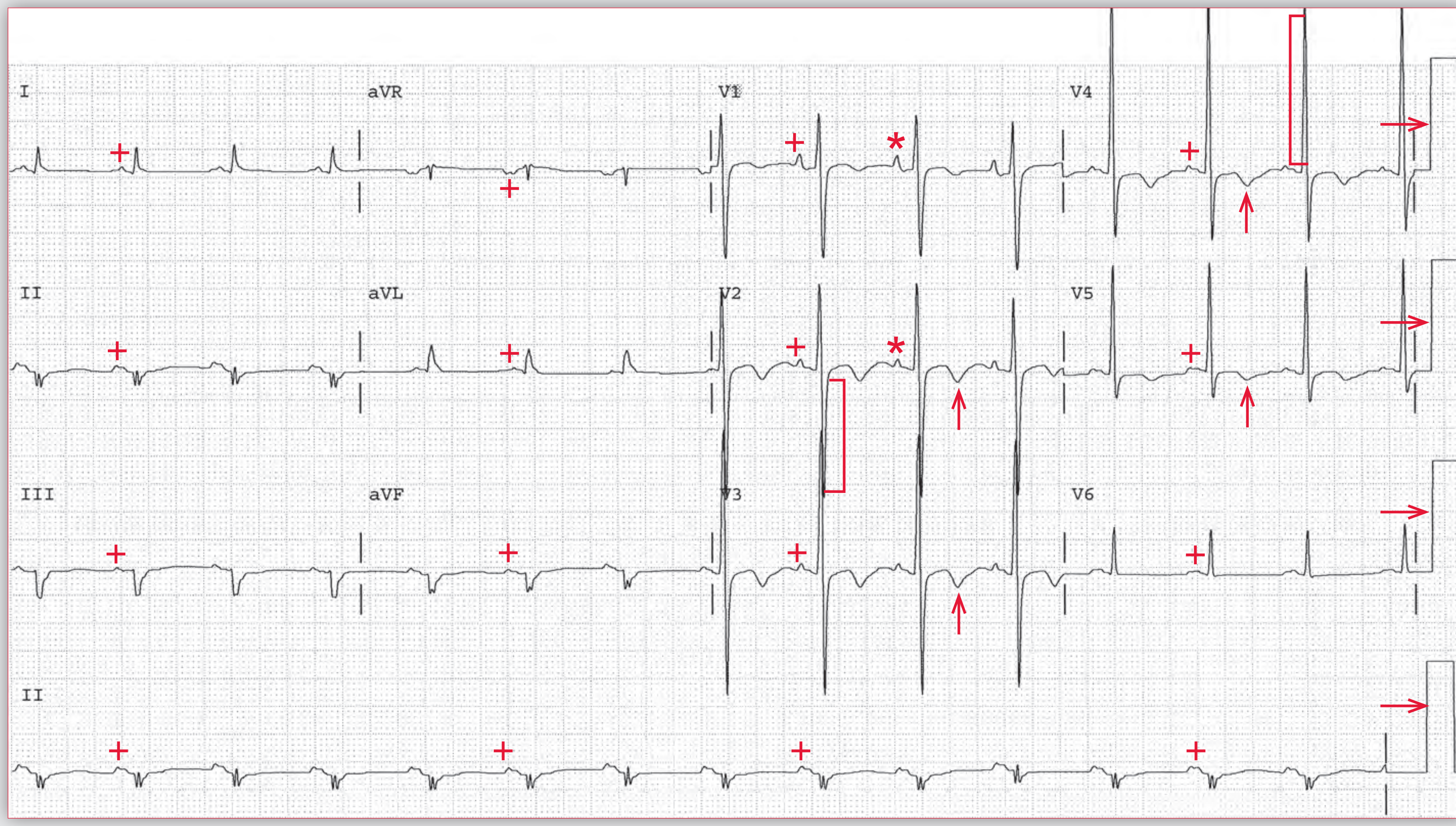


What abnormalities are noted on the initial tracing that lead to the house officer's suggestion of hypertrophic myopathies?

What maneuver did the attending physician perform to obtain ECG 45B and arrive at a different set of pathologic etiologies?

ECG 45B



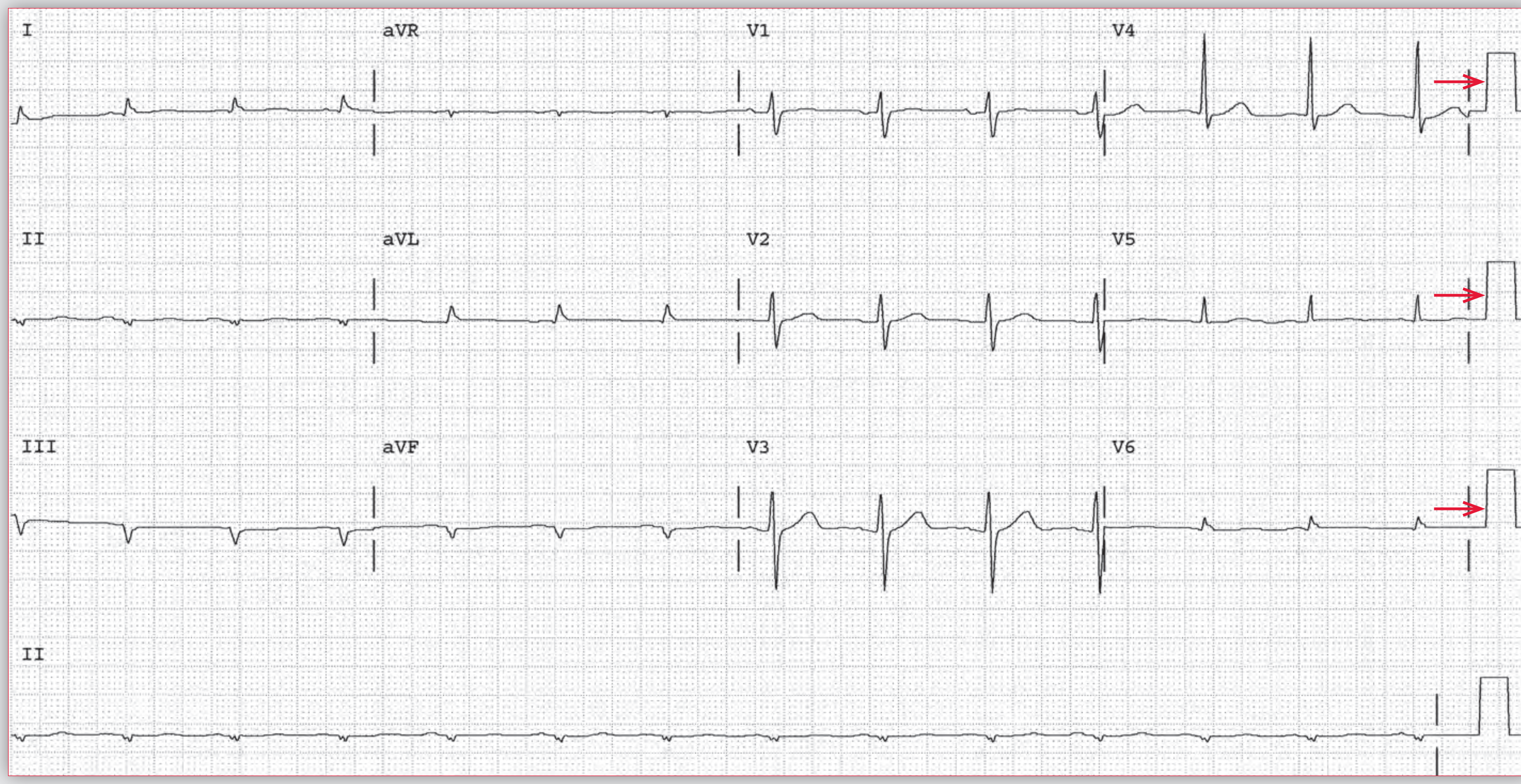


ECG 45A Analysis: Normal sinus rhythm, leftward axis, old inferior wall myocardial infarction, ECG recorded at double standard

ECG 45A shows there is a regular rhythm at a rate of 86 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this a normal sinus rhythm. The P wave in leads V1–V2 is positive and tall (*), suggesting right atrial hypertrophy or right atrial abnormality.

The QRS complex duration is normal (0.08 sec). The axis is extremely leftward between –30° and –90° (positive QRS in lead I and negative in leads II and aVF). As there are QS complexes in leads II and aVF, the extreme left axis is the result of an old inferior wall myocardial infarction. The QT/QTc intervals are normal (360/430 msec). There is low voltage in the limb leads (QRS complex < 5 mm in each lead). However, the voltage in the precordial leads is very high, with an R-wave amplitude in lead V4 = 30 mm ([ ) and the S wave in lead V2 = 22 mm ( ]). These meet the criteria for left ventricular hypertrophy (S + R in any two precordial leads ≥ 35 mm), associated with ST-T wave changes (↑) noted in leads V2–V5. However, it should be noted that this ECG was recorded at double standard (→), *ie*, 1 mV = 20 mm (20 boxes). Hence the amplitude of all the waveforms is twice normal. When adjusting for the double standardization, there is in fact no left ventricular hypertrophy, the P wave in lead V1 is normal, but there is still very low voltage in the limb leads.

continues



ECG 45B Analysis: Normal sinus rhythm, low limb lead voltage, leftward axis, old inferior wall myocardial infarction, ECG recorded at normal standardization

ECG 45B is from the same patient as ECG 45A, recorded at normal standardization (→) (1 mV = 10 mm or boxes). The rhythm and intervals are the same as in ECG 45A. However, the QRS amplitude in the precordial leads is now normal, and there is no evidence for left ventricular hypertrophy. The QRS voltage in the limb leads is very low.

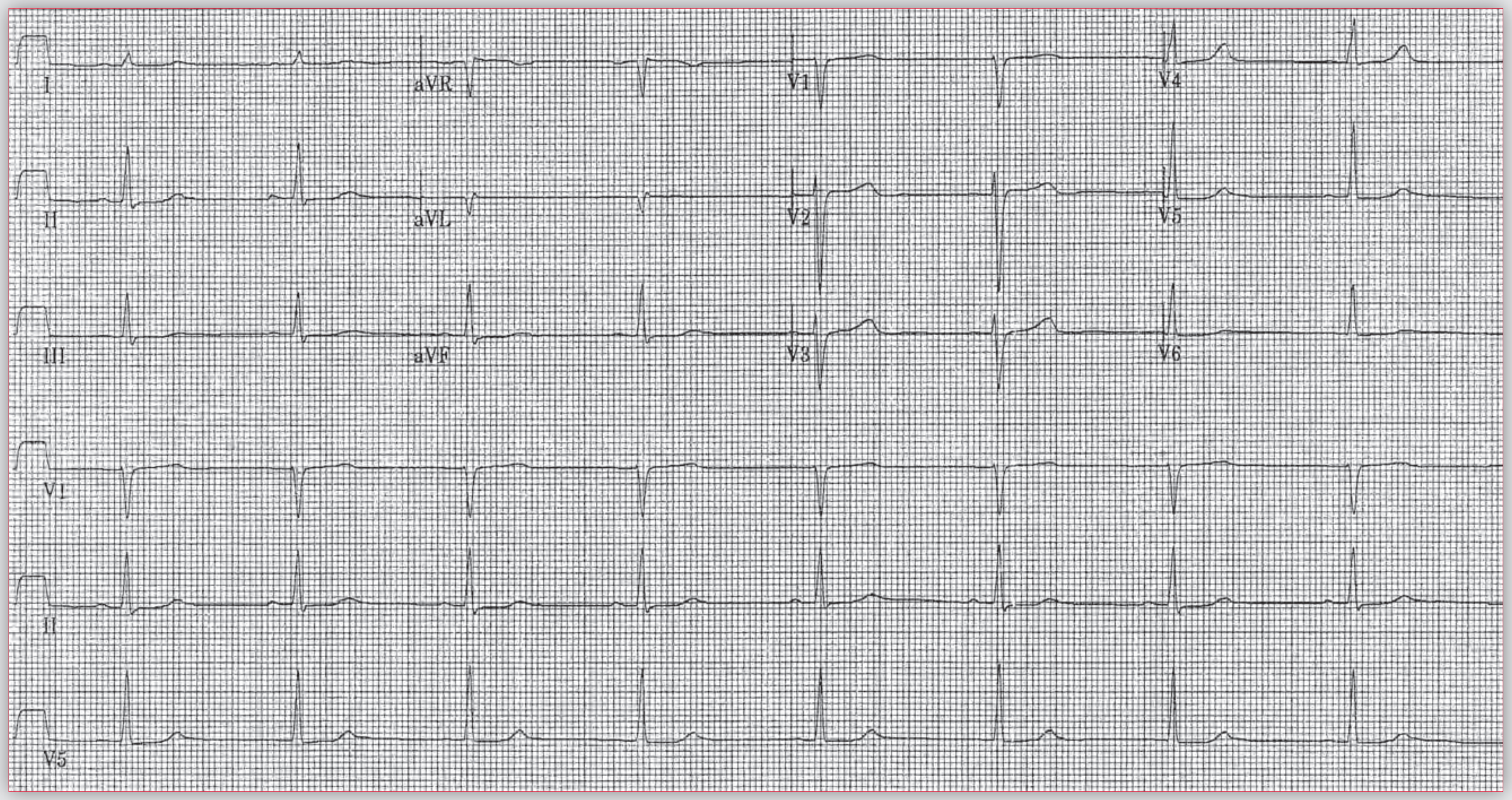
Low voltage has a number of etiologies that results in a reduction of transmission of electrical activity to the surface of the body, including obesity, pulmonary disease (particularly COPD), pericardial thickening, or a pericardial effusion. It may also result from a reduction in electrical activity generation as a result of loss of myocardium, such as occurs with an infiltrative cardiomyopathy or diffuse fibrosis due to significant myocardial infarction or a nonischemic dilated cardiomyopathy.

An ECG is occasionally recorded using double standard when the waveforms are small and not well seen or when looking for specific waveforms, for example P waves during a tachycardia. It is important to look at the standardization before interpreting the ECG so as to avoid misinterpreting the amplitude of waveforms, particularly the QRS complex. ■

Core Case 46

A medical student brings her attending physician the ECG of a female patient she has admitted (ECG 46A). Much to her surprise, the attending states that the patient should have an echocardiogram because of

ECG 46A

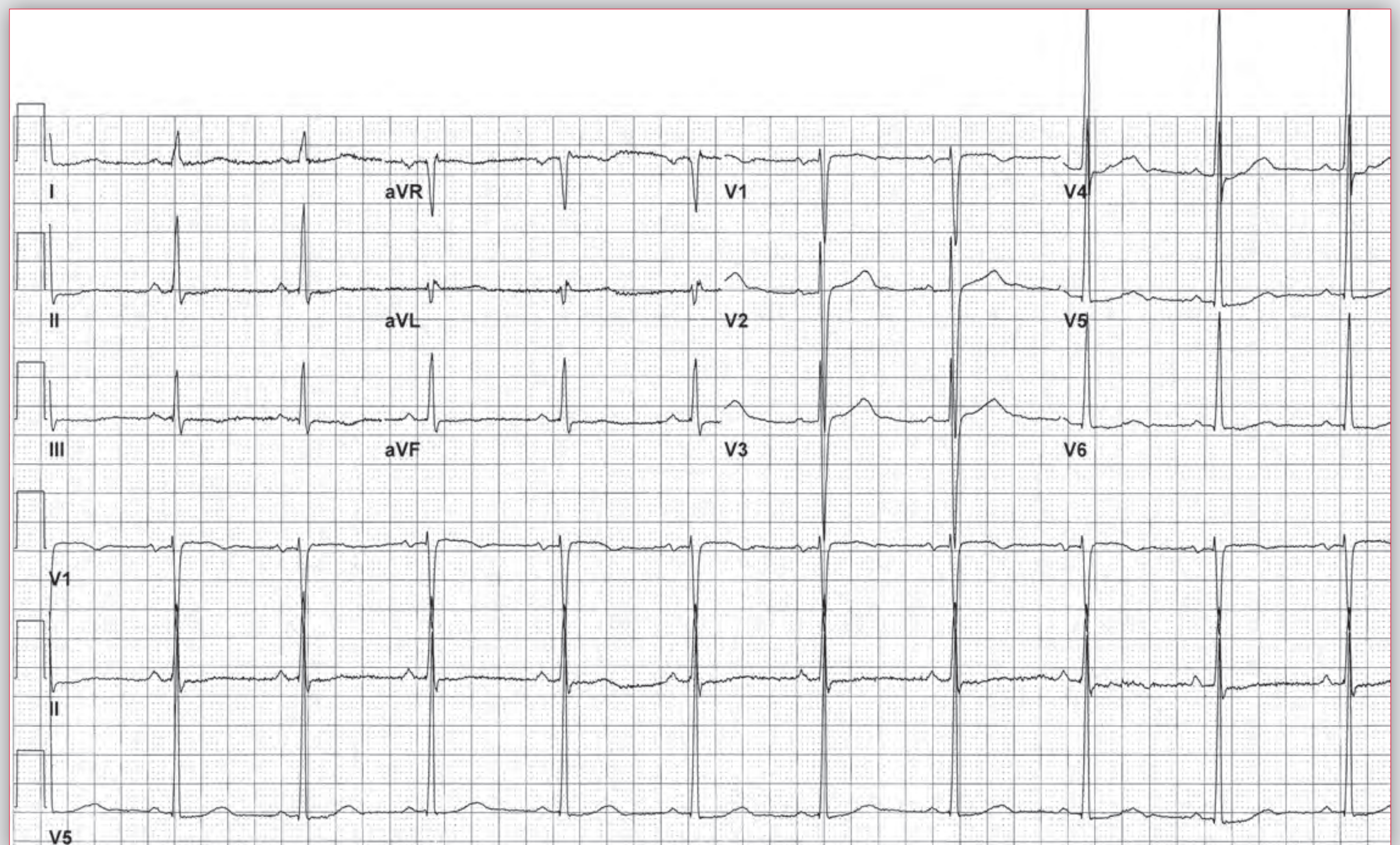


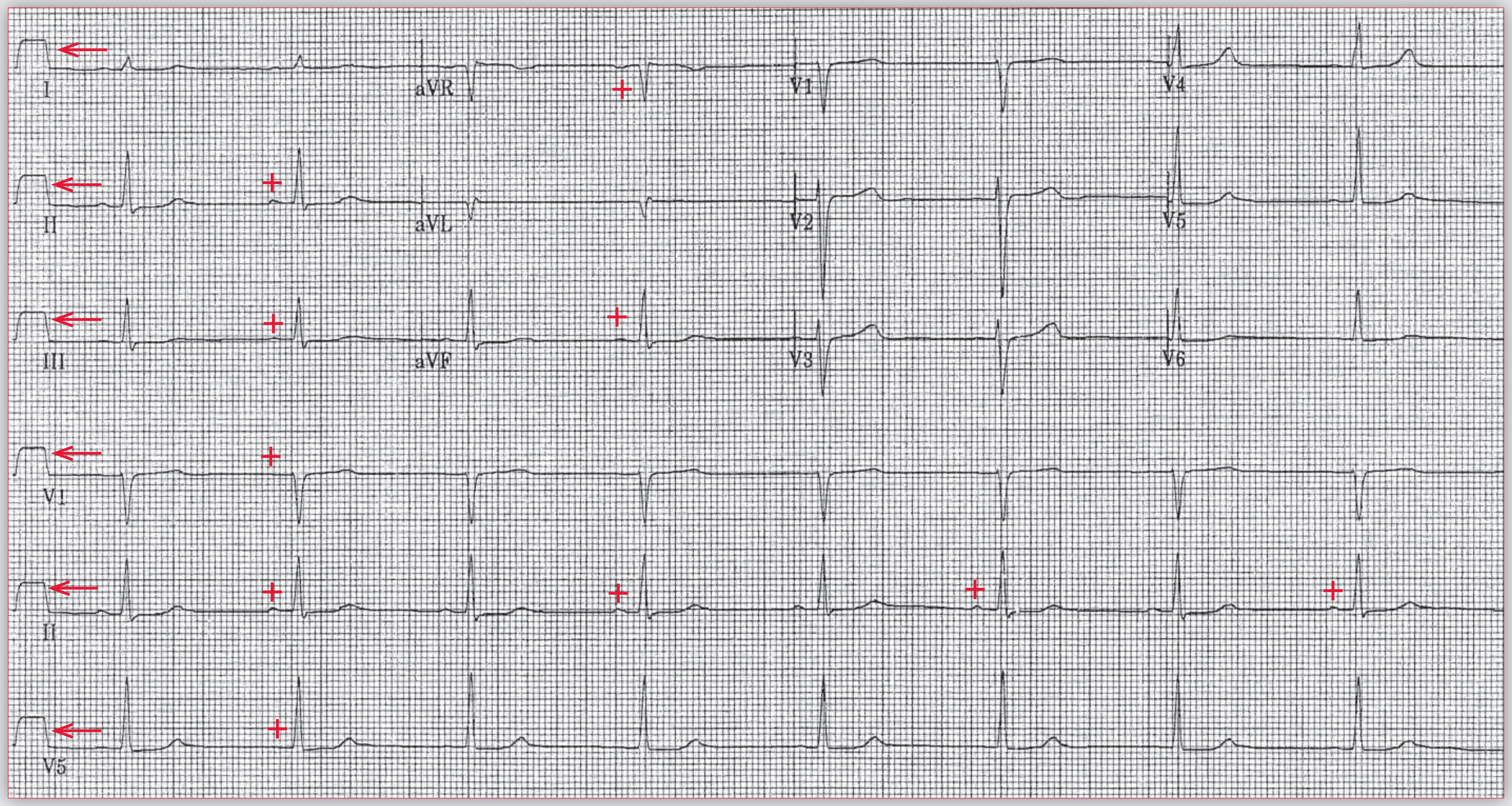
a myocardial abnormality based on the ECG.

Noticing the puzzled look on the student's face, the attending repeats the ECG (ECG 46B), upon interpretation of which the student realizes the basis for an echocardiogram.

What finding did the student miss from ECG 45A and why?

ECG 46B





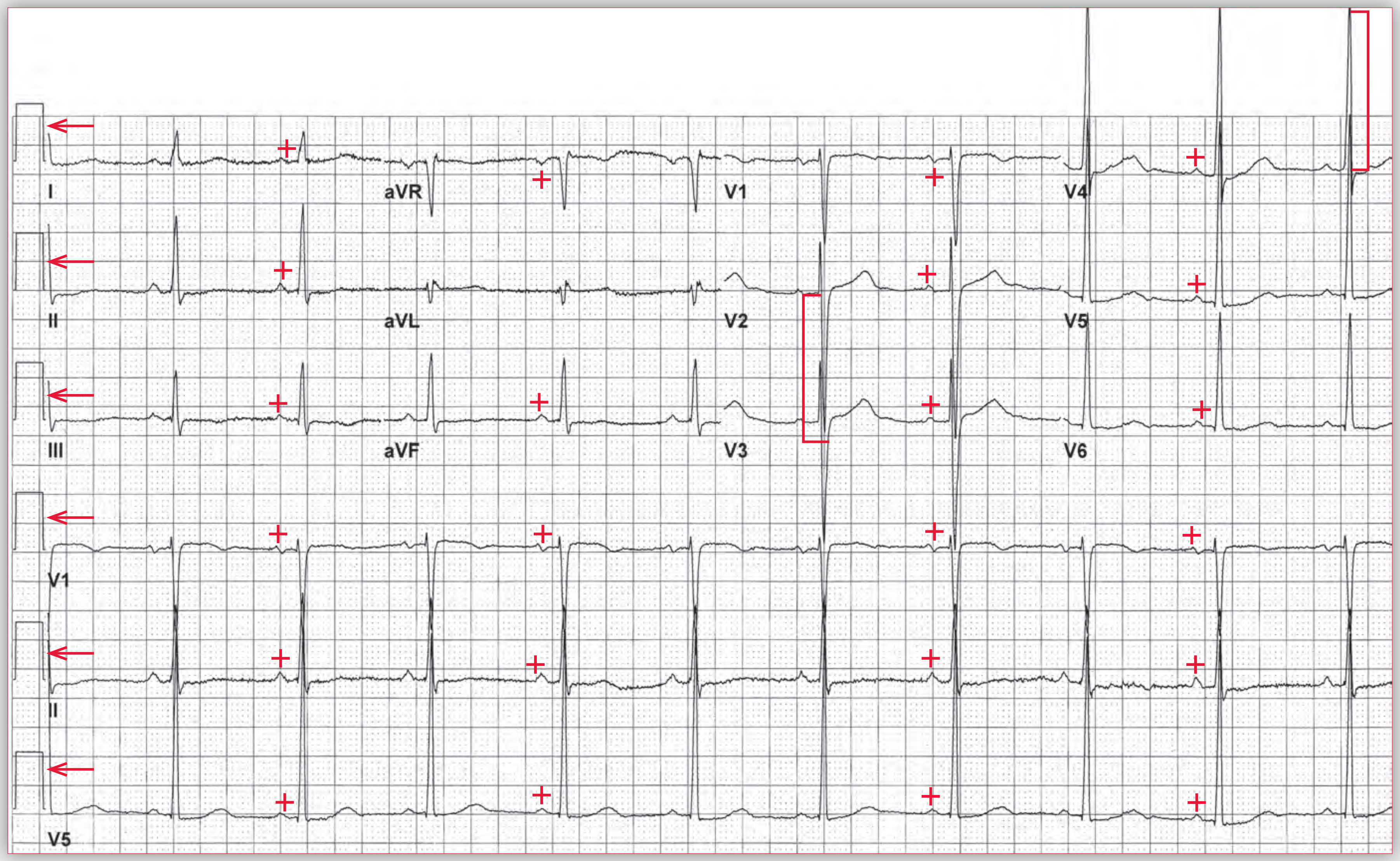
ECG 46A Analysis: Sinus bradycardia, ECG recorded at half standard

ECG 46A shows a regular rhythm at a rate of 50 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). Although the P waves have a low amplitude, they are positive in leads I, II, aVF, and V4 and negative in aVR. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec) and there is a normal morphology and normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (480/440 msec).

It should be noted that this ECG was recorded at half standard (←) (*ie*, 1 mv = 5 mm or boxes). Therefore, the amplitude of the waveforms is half of normal. Hence in lead V5, the R-wave amplitude is actually 26 mm and the S wave in lead V2 is actually 32 mm; this meets criteria for left ventricular hypertrophy.

continues



ECG 46B Analysis: Sinus bradycardia, left ventricular hypertrophy, ECG recorded at normal standardization

ECG 46B. This ECG is from the same patient as ECG 61A, recorded with normal standardization (←) (1 mV = 10 mm or boxes). The rhythm, rate, and intervals are the same as in ECG 61A. However, the P wave (**+**) amplitude is now normal. The QRS complex amplitude is increased; the R wave in lead V5 ( ]) is 30 mm and the S wave in lead V2 ([ ) is 25 mm, meeting criteria for left ventricular hypertrophy (*ie*, S V2 **+** R V5 ≥ 35 mm).

On occasion an ECG is recorded at half standard when the amplitude or height of the QRS complexes is very increased, causing the superimposition of waveforms of leads that are in the same column. ■

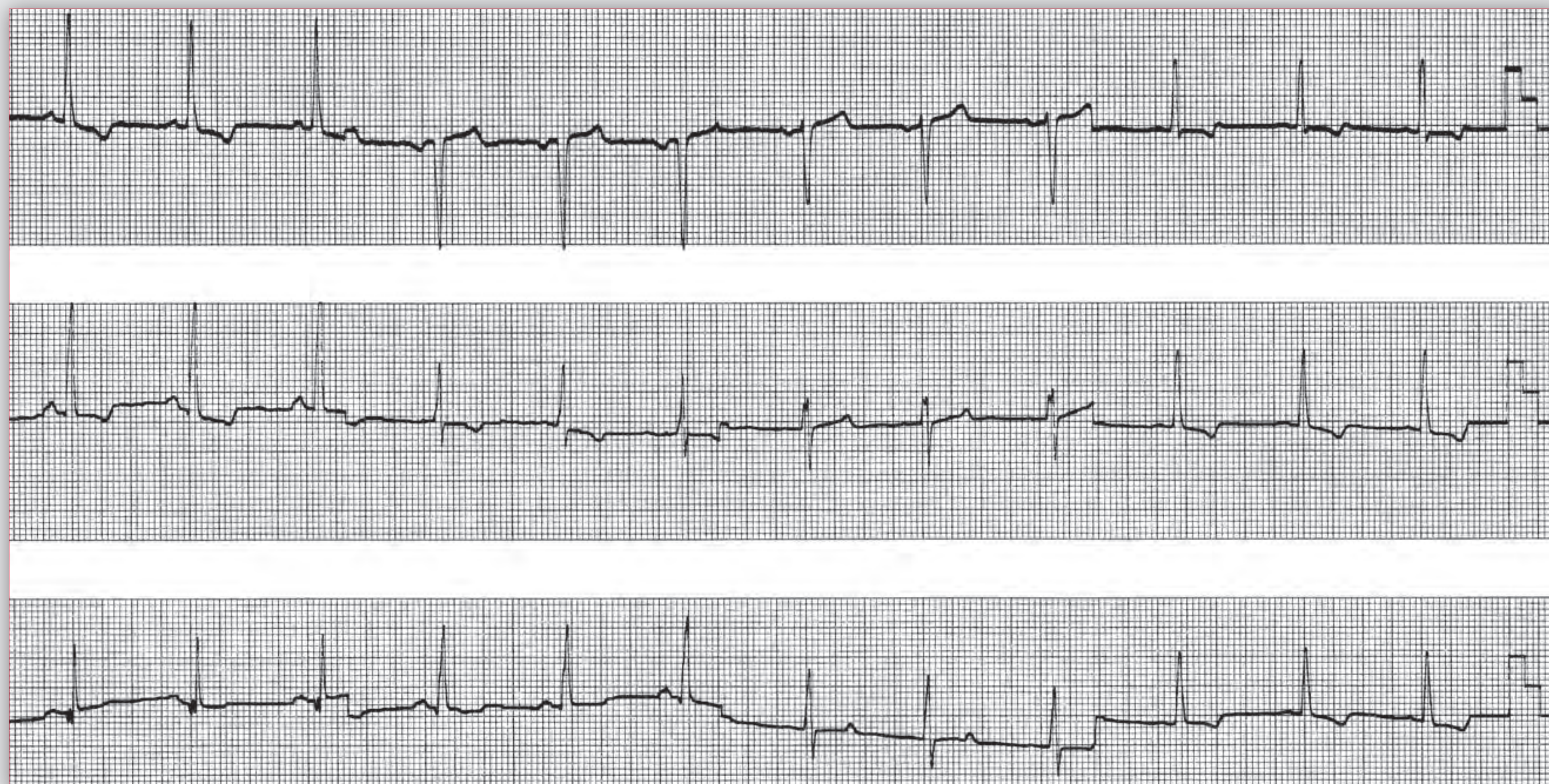
Notes

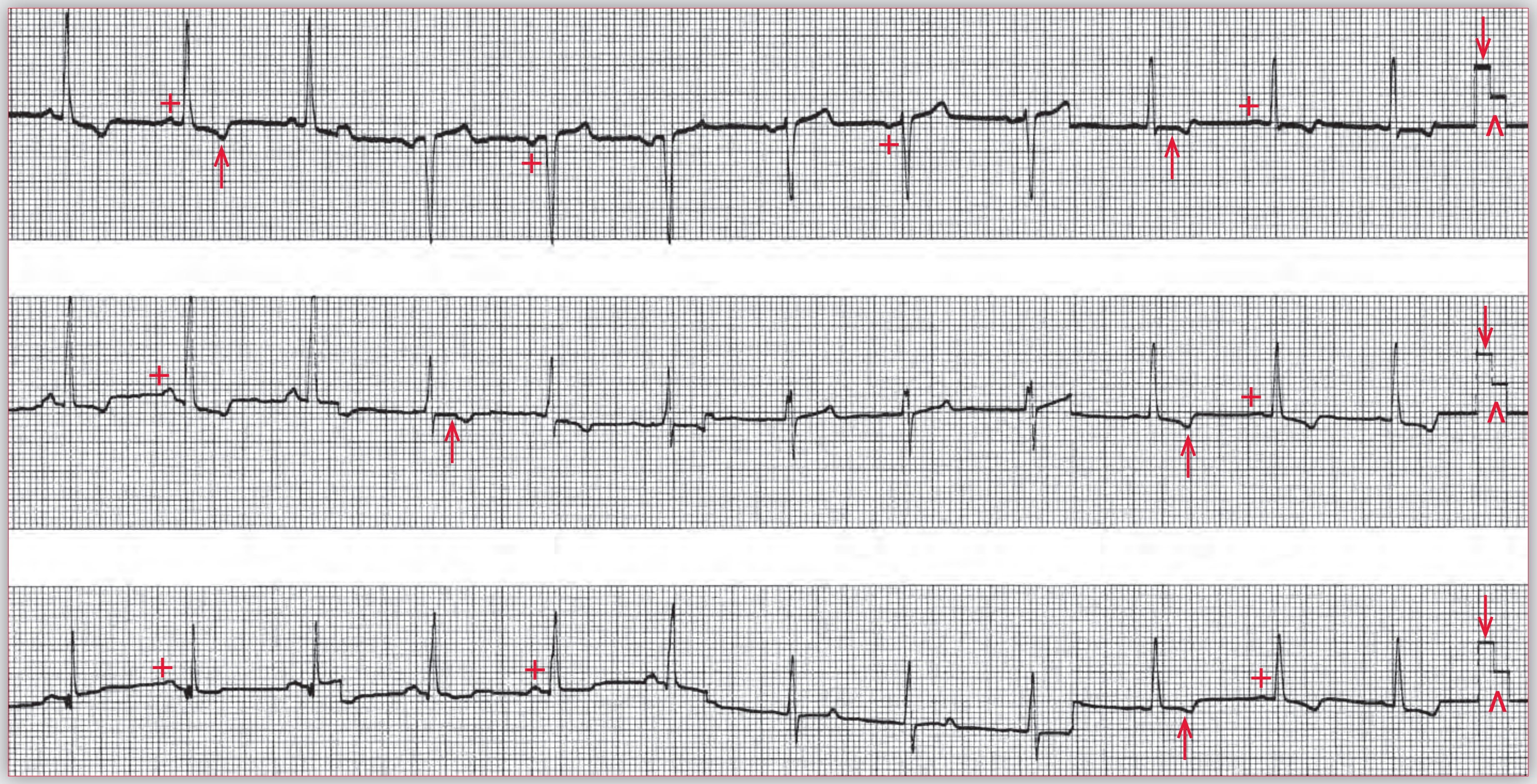
Core Case 47

A 56-year-old man with a history of severe hypertension that has been poorly controlled over the past 5 years presents to his physician with a history of chest pain that began the night before. Although the chest pains are sharp, localized and fleeting, the physician is concerned for myocardial ischemia. BP is 188/110, and this is similar to what was noted during his last visit. An ECG is obtained and is noted to be different from a previous ECG obtained 6 months ago.

What abnormalities are present?

Does this patient have left ventricular hypertrophy?





ECG 47 Analysis: Normal sinus rhythm, limb leads recorded normal standard, precordial leads recorded half standard, left ventricular hypertrophy with associated ST-T wave changes

There is a regular rhythm with a rate of 70 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4–V6 and negative in aVR. Hence this is a sinus rhythm.

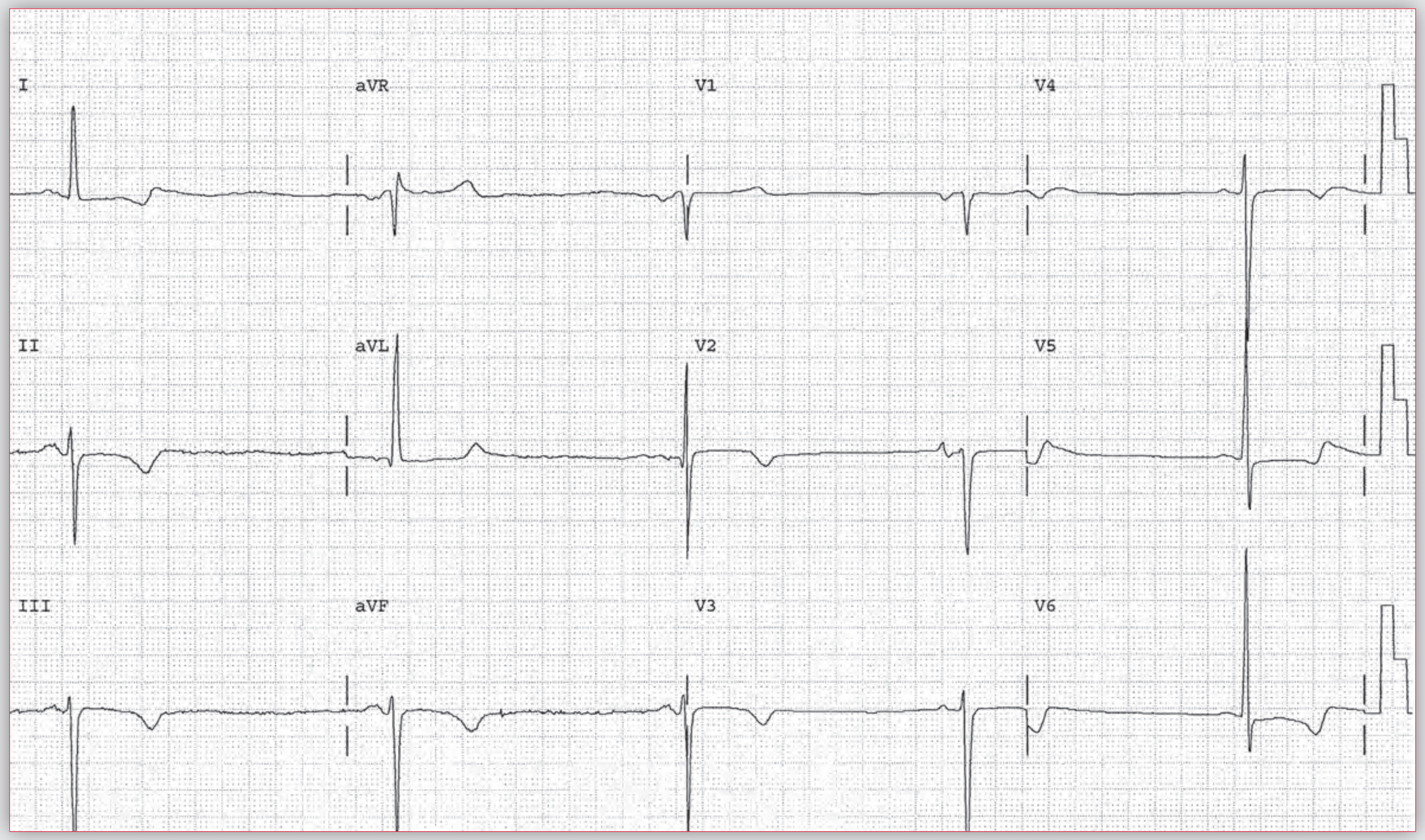
The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/410 msec). Although the QRS amplitude in the limb leads is tall (18 mm in lead I), the amplitude does not meet criteria for left ventricular hypertrophy (*ie*, R wave in any limb lead ≥ 20 mm). The amplitude of the QRS complexes in the precordial leads also does not meet criteria for left ventricular hypertrophy, although there are ST-T wave changes (↑) present that are often seen with left ventricular hypertrophy. Although the J point is normal, there is downsloping ST-segment depression, which is a pattern seen with subendocardial ischemia that is often present with left ventricular hypertrophy. However, in the absence of hypertrophy, there would be a concern for ischemia due to epicardial coronary artery disease. However, it should be noted that the limb leads were recorded at normal standardization (↓) (1 mV = 10 mm or boxes) while the precordial leads were recorded at half standard (^) (1 mV = 5 mm or boxes). Therefore, the amplitude of the R or S wave in the precordial lead needs to be doubled, *ie*, the amplitude of the R wave in lead V5 is 26 mm and the S wave in lead V2 is 14 mm. Hence criteria for left ventricular hypertrophy are met (*ie*, S V2 + R V5 ≥ 35 mm) and it is likely that the ST-segment changes are the result of left ventricular hypertrophy. The most common reason for left ventricular hypertrophy is systemic hypertension. This patient has had poorly controlled blood pressure, and it is likely that there has been some progression of hypertrophy as well as progression of ST-segment changes as a result.

Although many of the ECG machines will indicate that the ECG tracing was recorded at half or double standardization, the standardization icon should be quickly looked at to be certain. ■

Core Case 48

A 59-year-old woman with a history of hypertension presents to his primary care physician for a routine examination. She has no complaints and has not noted any new symptoms. She states that her blood pressure measurements

ECG 48A



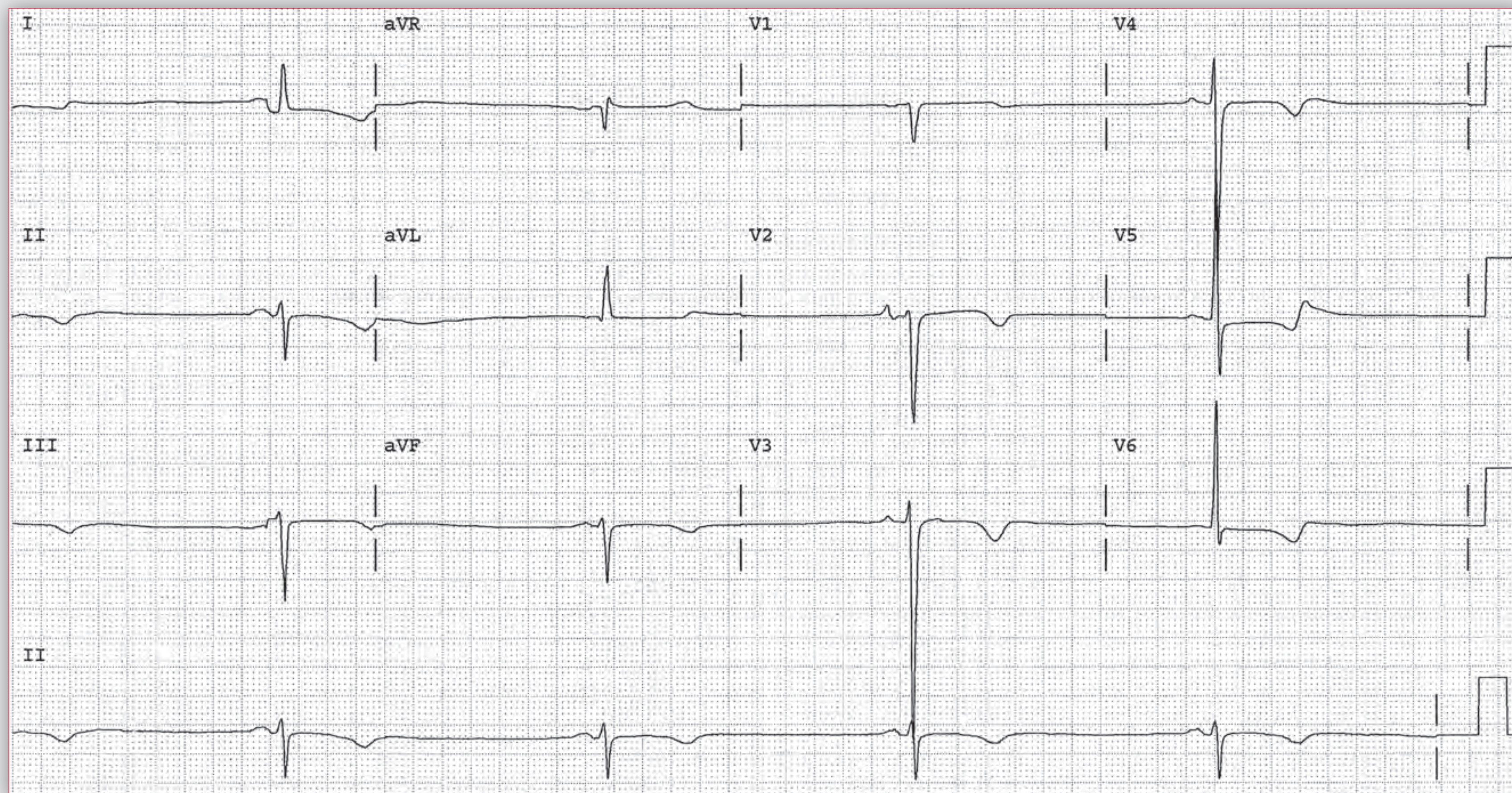
at home have been normal. Her medications include a β-blocker (Toprol 200 mg/day), hydrochlorthiazide (12.5 mg/day) and diltiazem (240 mg/day). A routine ECG is obtained (ECG 48A) and compared to the previous ECG (ECG 48B).

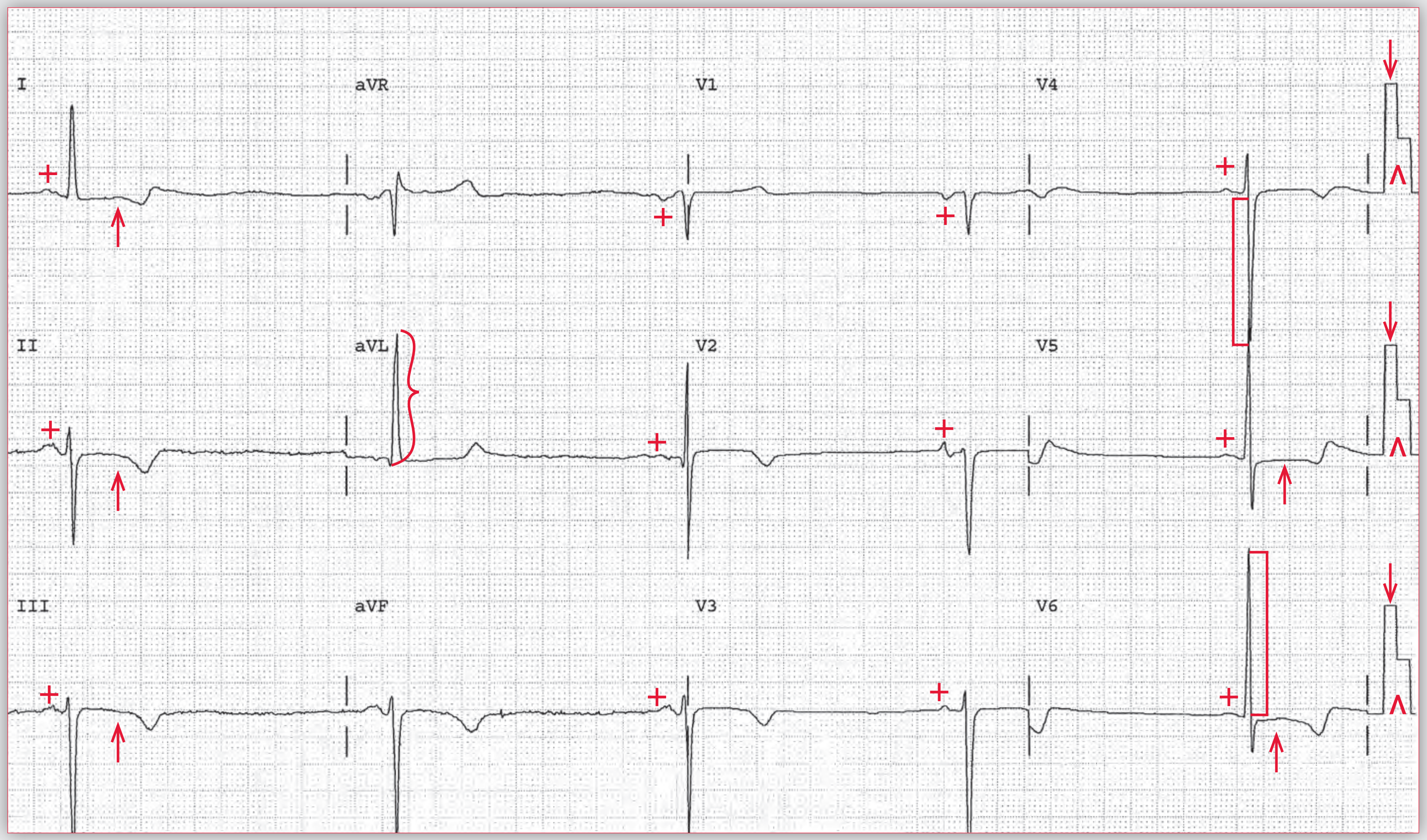
Are the ECGs the same?

If not, what is the difference?

What accounts for the differences?

ECG 48B





ECG 48A Analysis: Sinus bradycardia, left ventricular hypertrophy with associated ST-T wave abnormalities, left atrial abnormality, limb leads recorded at double standard, precordial leads recorded at normal standard

ECG 48A shows there is a regular rhythm at a rate of 30 bpm. Note is made that this ECG was recorded at normal speed of 25 mm/sec. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Therefore, this is a sinus bradycardia. The P wave is broad and notched in leads II and aVF and negative in lead V1, consistent with left atrial hypertrophy.

The QRS complex duration is normal (0.10 sec) and there is a normal morphology. The axis is extremely leftward between –30° and –90° (positive in lead I and negative in leads II and aVF with an rS morphology). This is called a left anterior fascicular block. The amplitude of the QRS complex in lead aVL is 23 mm (}), meeting one of the criteria for left ventricular hypertrophy, *ie*, R wave > 11 mm or > 18 mm in the presence of a left axis. However, it is noted that the limb leads were recorded at double standard (↓), *ie*, 1 mV = 20 mm or boxes. Therefore, the actual amplitude of the R wave in aVL is 12 mm.

The precordial leads were recorded at normal standardization (^) (1 mv = 10 mm or boxes). The R-wave amplitude in V6 is 30 mm ([ ) and the depth of the S wave in lead V4 is 25 mm ( ]). Hence a criterion for left ventricular hypertrophy is met in the precordial leads (*ie*, the S wave and R wave in any two precordial leads ≥ 35 mm). There are associated ST-T wave changes (↑) noted in the limb leads and the precordial leads.

The QT/QTc intervals are slightly prolonged (660/460 msec), but the QTc interval is probably normal for a female.

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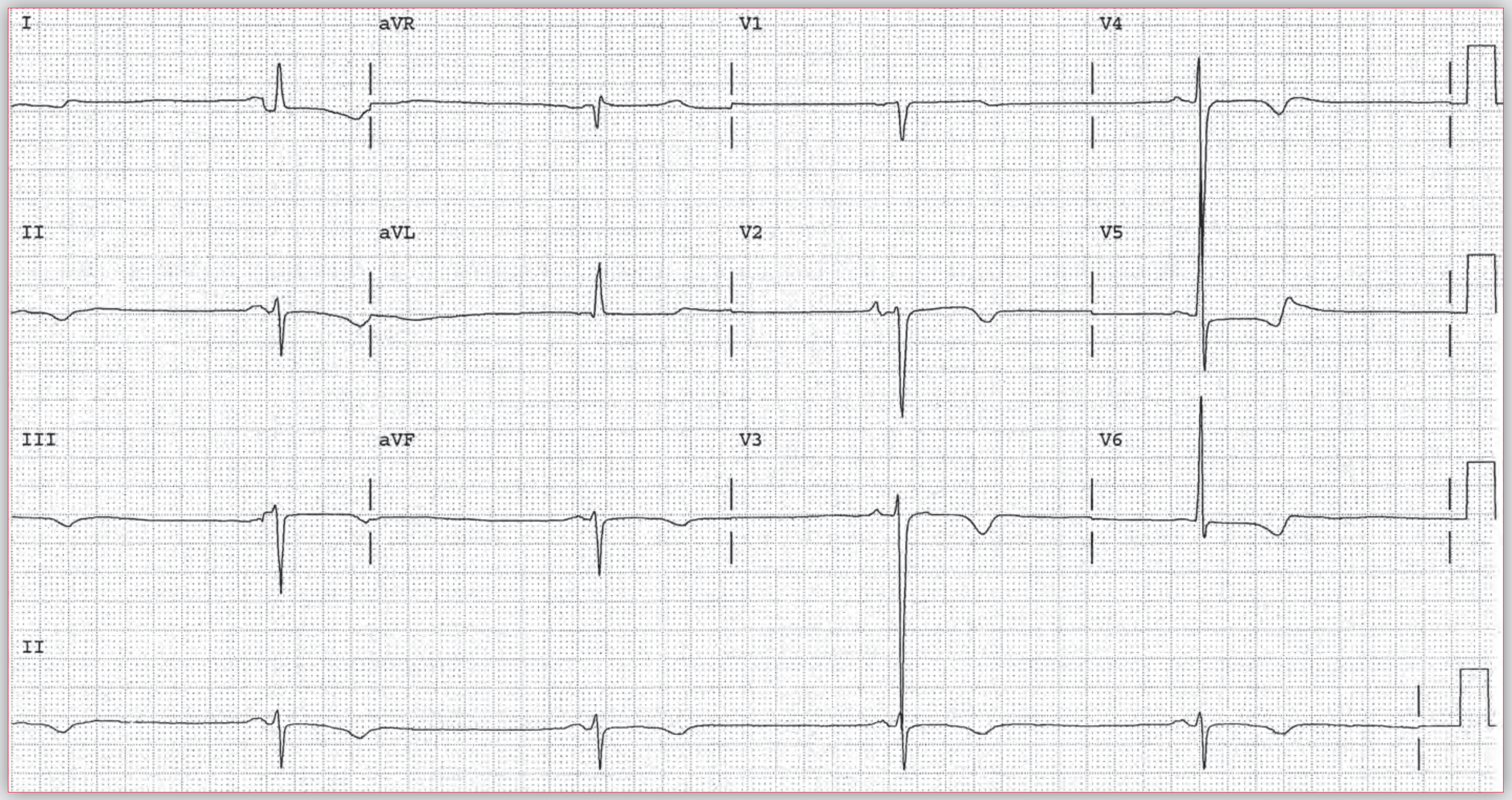


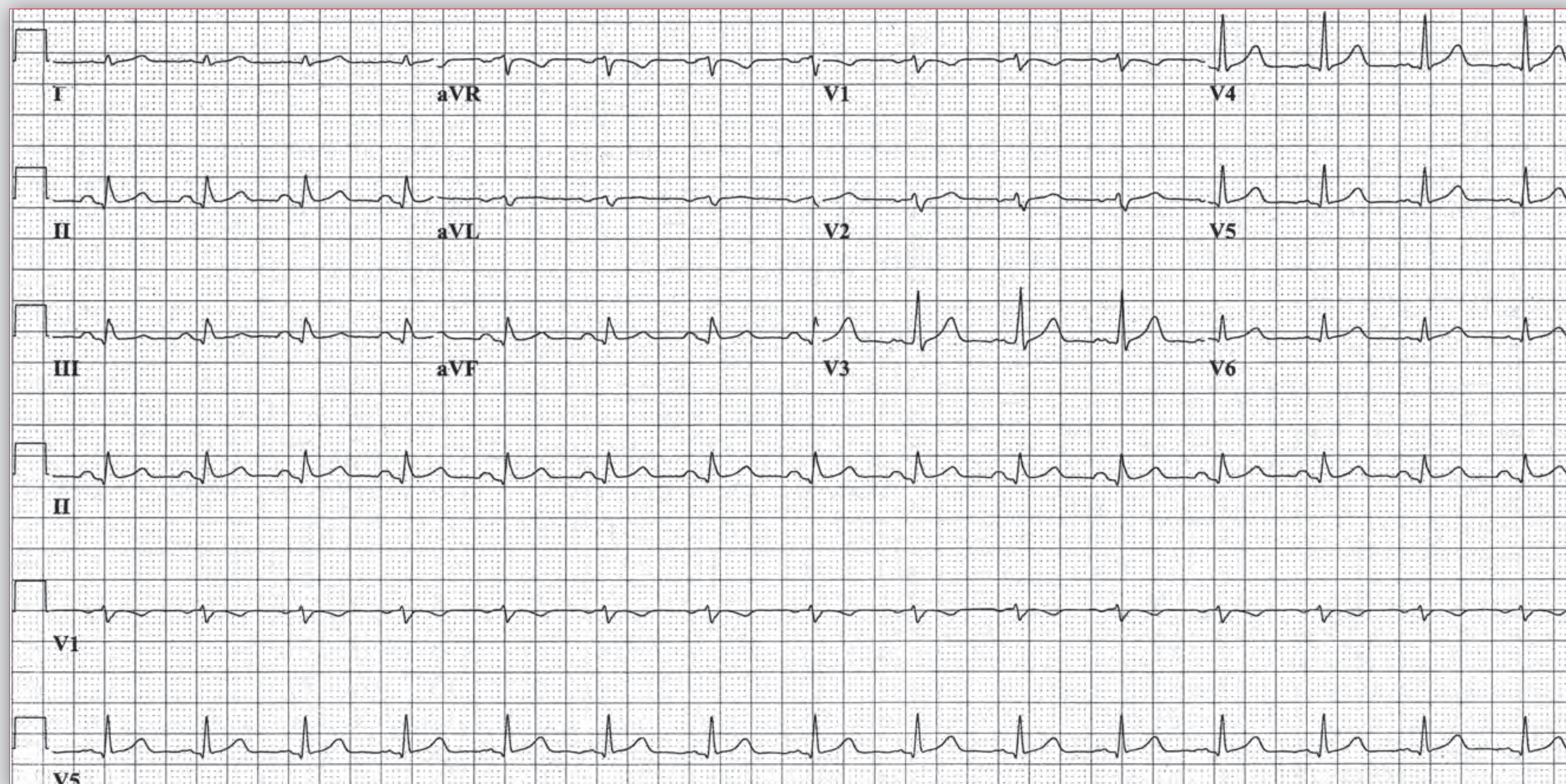
ECG 48B Analysis: Sinus bradycardia, left ventricular hypertrophy with ST-T wave changes, left atrial abnormality

ECG 48B is from the same patient. This entire ECG was recorded at normal standardization. The heart rate, rhythm, axis and intervals are the same as in ECG 48A. The P wave and QRS complex morphologies are also the same. However, now that the limb leads are also recorded at normal standardization (1 mv = 10 mm or boxes) it can be seen that the amplitude or voltage of the QRS complex in the limb leads is normal. ■

Core Case 49

A 56-year-old airline pilot without any previous medical or cardiac history presents to his internist for a yearly routine physical examination mandated by the Department of Transportation. He has no complaints. Physical examination is unremarkable. An ECG is obtained (ECG 49A), and the physician is concerned because of a change from his ECG 1 year ago (ECG 49B).

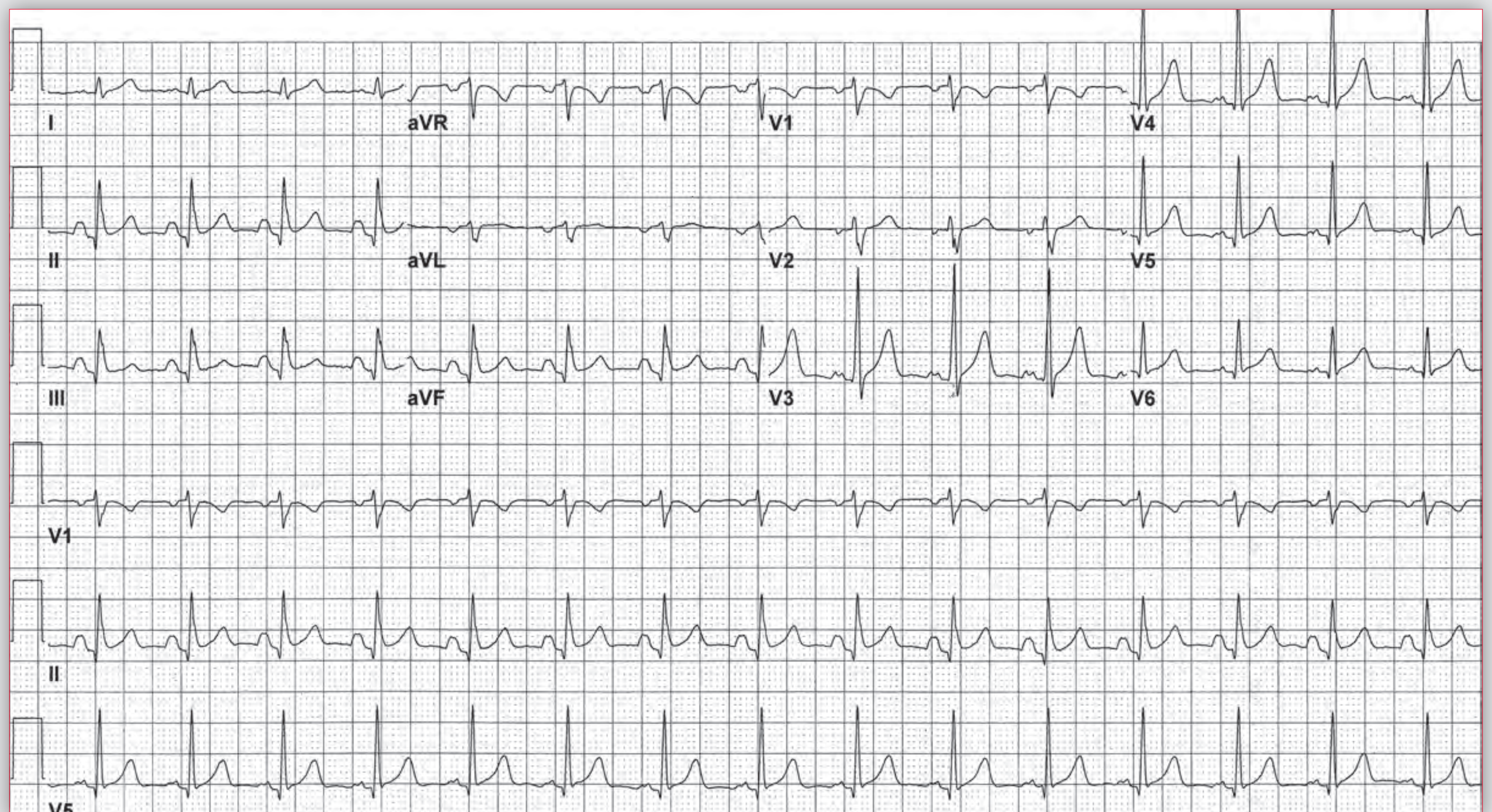
ECG 49A

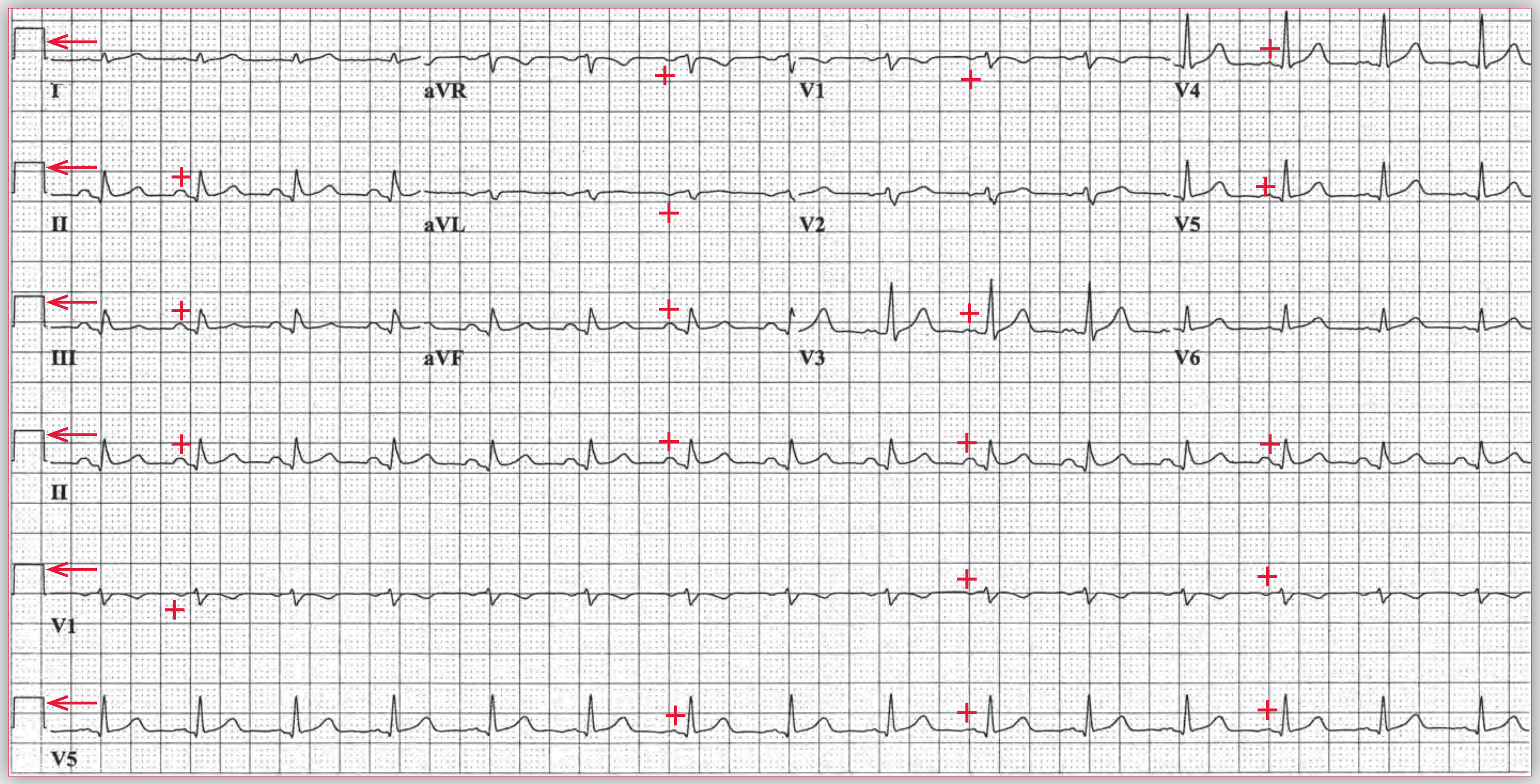


What is the finding of concern?

What is the reason for the change?

ECG 49B



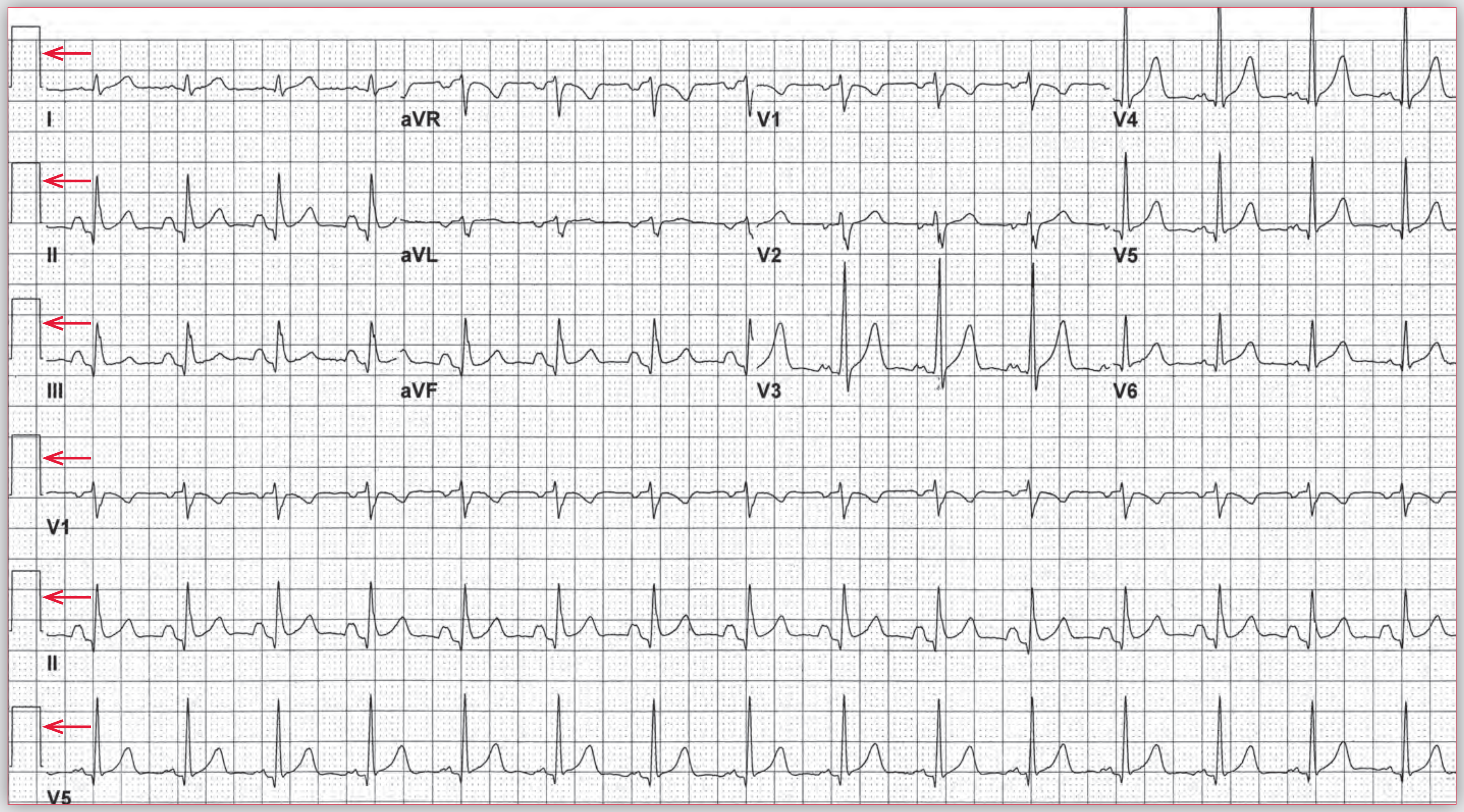


ECG 49A Analysis: Normal sinus rhythm, low voltage, ECG recorded at half standard

ECG 49A shows there is a sinus rhythm with a rate of 90 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V5–V6 and negative in aVR. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/390 msec). There is low voltage in both the limb leads (QRS < 5 mm in each lead) and precordial leads (QRS < 10 mm in each limb). However, it should be noted that the entire ECG was recorded at half standard (←) (1 mV = 5 mm). Hence the amplitude of the QRS complex is twice what is measured. Thus the QRS complexes have a normal voltage.

continues



ECG 49B Analysis: Normal sinus rhythm, normal standardization

ECG 49B is from the patient with ECG 49A, and it was recorded using normal standardization (←) (1 mv = 10 mm or boxes). The rate, rhythm, intervals, and P wave and QRS morphology are the same as on ECG 49A. The QRS amplitude in all the leads is normal.

On occasion the ECG may be recorded at half standard when the amplitude of the QRS complex is very tall and the complexes are overlapping. However, recording the ECG at half standard may not be intentional, as appears to be what occurred in this patient. Many of the ECG machines will indicate that the ECG was recorded at half standard. However, it is important that the standardization icon be looked at to establish how the ECG was recorded. ■

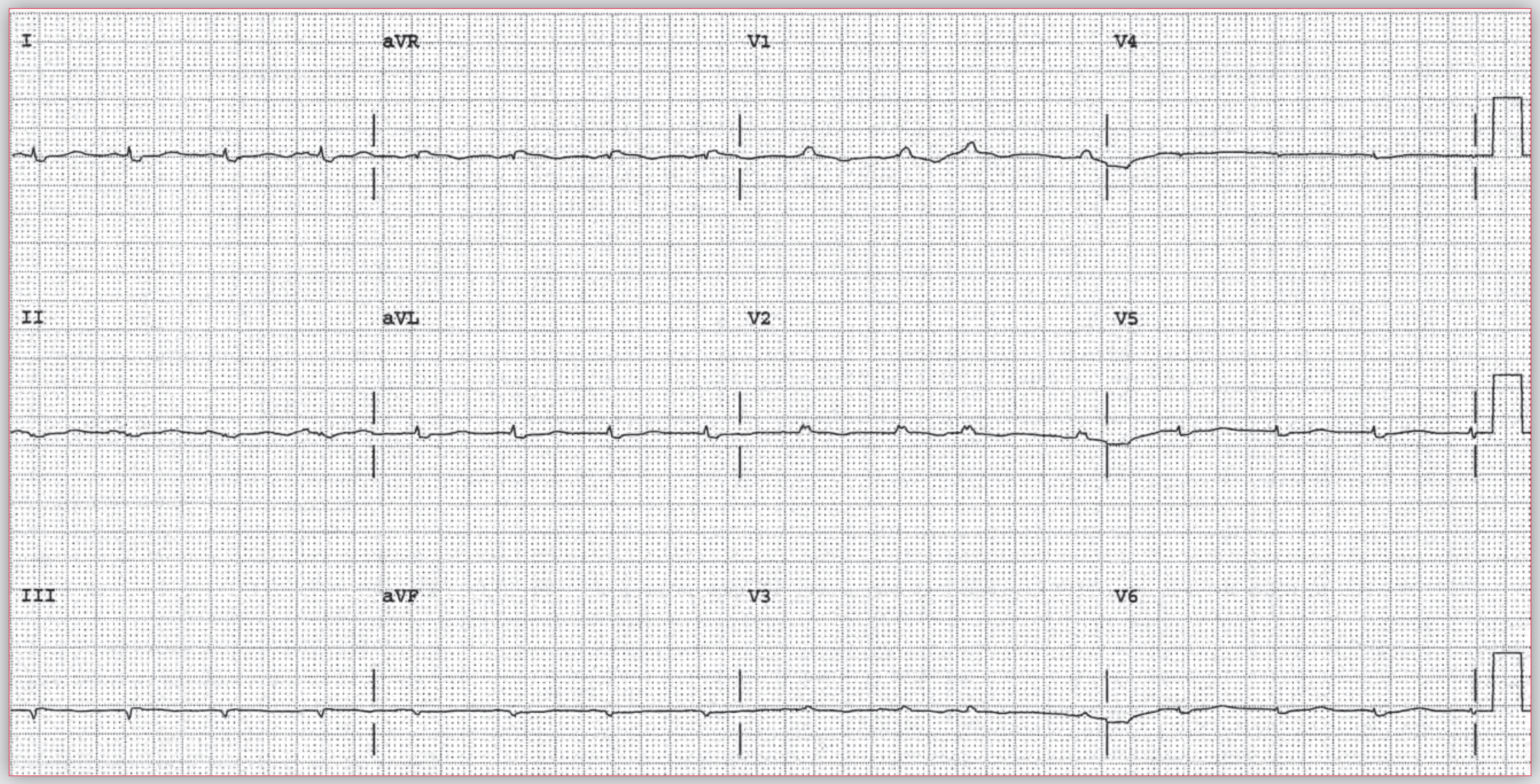
Notes

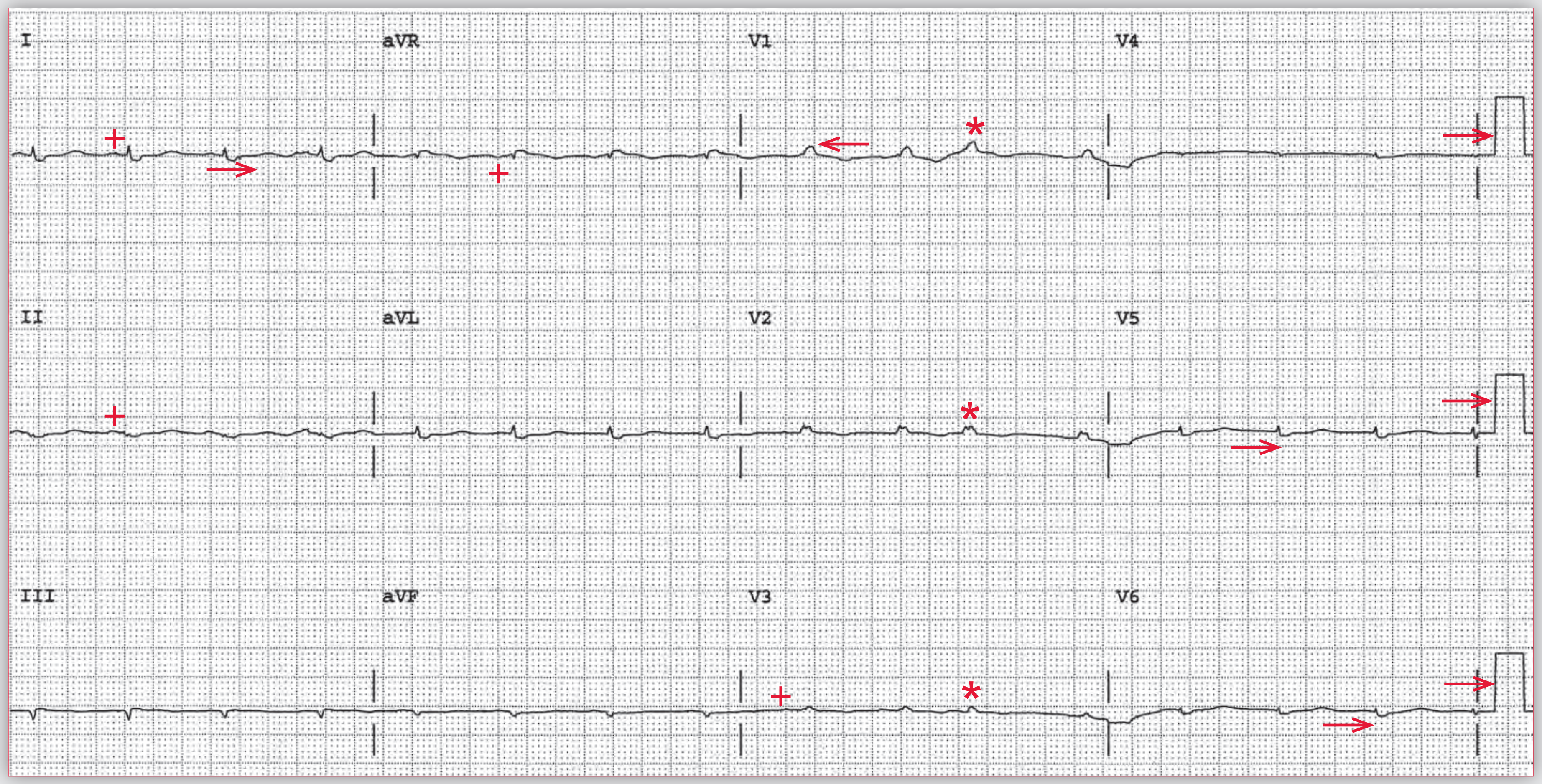
Core Case 50

A 70-year-old male with a known cardiomyopathy and left ventricular ejection fraction (LVEF) of 55% presents to the emergency department with complaints of severe shortness of breath and new-onset peripheral edema. Physical examination is remarkable for bilateral rales, bilateral peripheral edema, neck vein distention, and hepatomegaly. Chest x-ray shows evidence of mild vascular congestion. He is admitted to the hospital for heart failure and begun on intravenous diuresis. An ECG is obtained.

What does this show?

What type of cardiomyopathy is likely present?





ECG 50 Analysis: Low voltage in limb leads and precordial leads (recorded at normal standardization). Sinus rhythm, premature atrial complex, leftward axis, right bundle branch block

There is a regular rhythm with a rate of 92 bpm. P waves (+) can be seen before each QRS complex, although they are of low amplitude. The P waves are best seen in leads I, II, and aVR. There is a stable PR interval (0.14 sec).

The QRS complex duration is prolonged (0.12 sec). There is a broad R wave in lead V1 (←) and broad S wave in leads I and V5–V6 (→), consistent with a right bundle branch block morphology. The axis is leftward, possibly extremely leftward (*ie*, between –30° and –90°). There are two causes for an extreme left axis, *ie*, an old inferior wall myocardial infarction with a deep Q wave in leads II and aVF or a left anterior fascicular block with a rS QRS morphology in leads II and aVF. However, the etiology is difficult to establish as the QRS voltage is extremely low in all the limb leads (QRS < 5mm in each lead) and precordial leads (QRS < 10 mm in each lead). It should be noted that the ECG was recorded at normal standardization (→) *ie*, 1 mV equals 10 mm or 10 little boxes in amplitude. Hence this is true low voltage.

The QT/QTc intervals are prolonged (400/495 msec) even when the prolonged QRS complex duration is considered (380/470 sec). However, this may not be accurate considering the low voltage and small complexes. There is one QRS complex that is early (*). It has the same QRS morphology as the other complexes. Although it is not certain if it is preceded by the P wave, it is likely a premature atrial complex.

Low voltage, which reflects a reduction in the amount of electrical current reaching the surface of the body to be recorded by the electrodes, may be seen with extensive myocardial disease or infiltration (*eg*, amyloid), with a pericardial effusion, with a thick pericardium, in the setting of severe lung disease (COPD) or obesity. As the patient has a known cardiomyopathy with preserved LVEF, it is most likely that there is an infiltrative cause for the cardiomyopathy.

A particular concern in this patient would be amyloid heart disease. ■

Notes

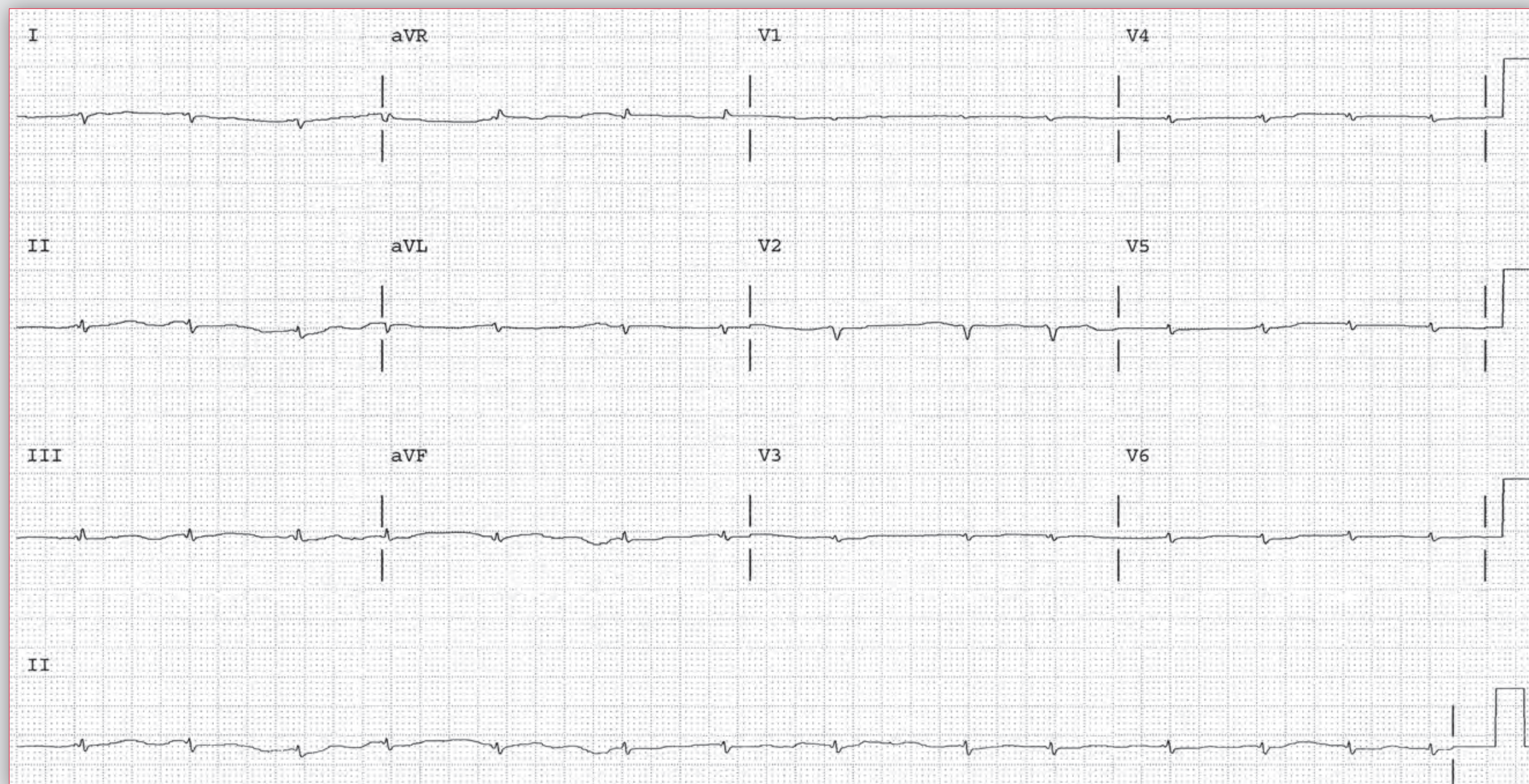
Core Case 51

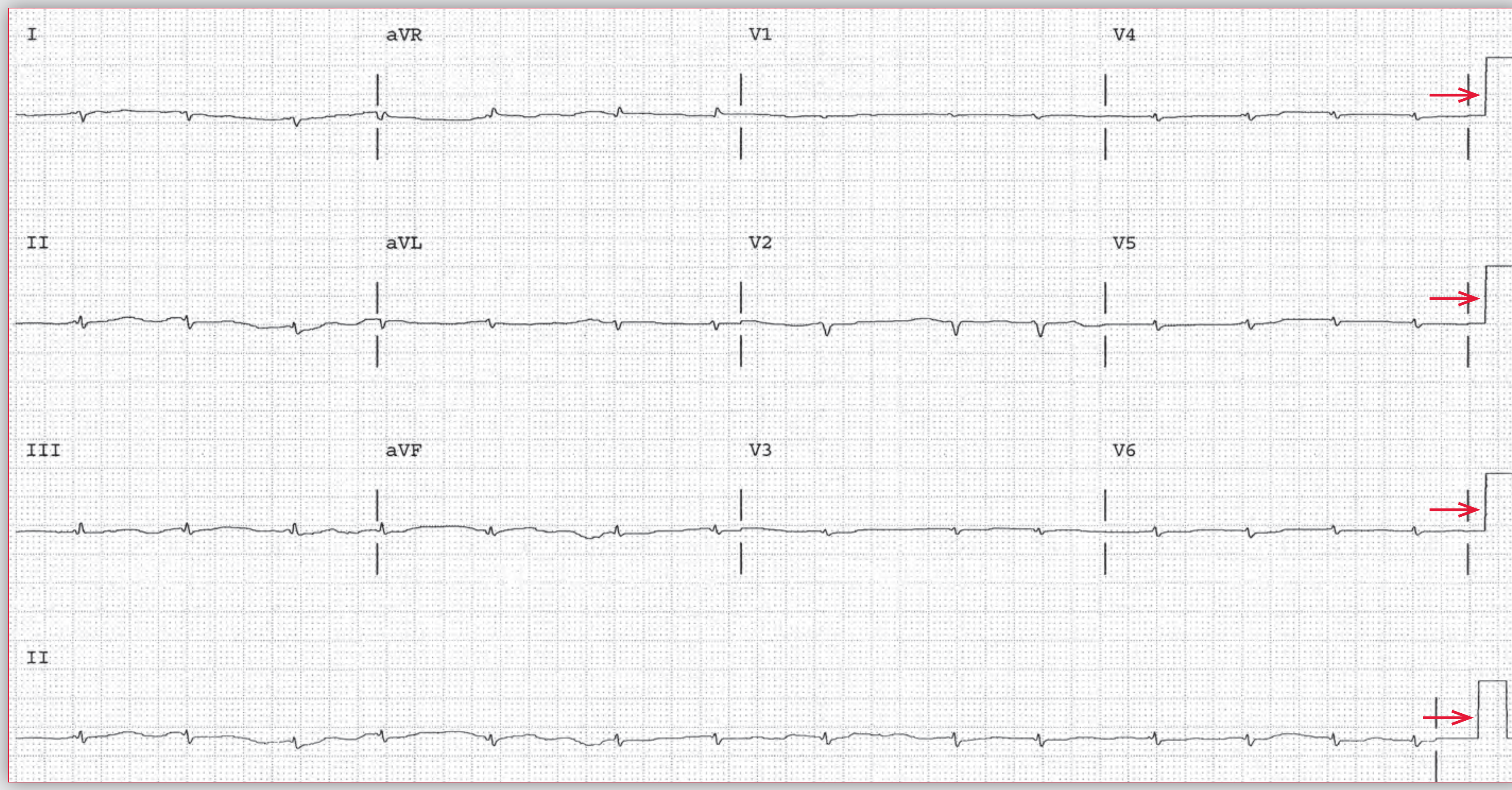
A 17-year-old female patient is admitted to an intensive care unit (ICU). She presented to the hospital emergency department 3 days after onset of an upper respiratory infection with fever. She rapidly displayed signs of shock and is transferred from the emergency department to the ICU. She does not carry any known diagnoses nor take any medications.

On arrival to the ICU, she is intubated and sedated. Her pulse is normal and her blood pressure is 94/40 with the assistance of two pressor agents. Her lungs are clear. Heart sounds are regular but difficult to hear under the ventilator sounds. Her abdomen is benign. Her extremities are cool to the touch and nonedematous. Peripheral pulses are thready.

An ECG is obtained.

While awaiting invasive hemodynamic data to better understand the etiology of her condition, to what diagnosis do the history and ECG point?





ECG 51 Analysis: Low voltage limb and precordial leads, atrial fibrillation, left posterior fascicular block

There is an irregularly irregular rhythm with a rate of 84 bpm. There are no obvious P waves seen, although the waveforms are of low amplitude. Hence this is probably atrial fibrillation.

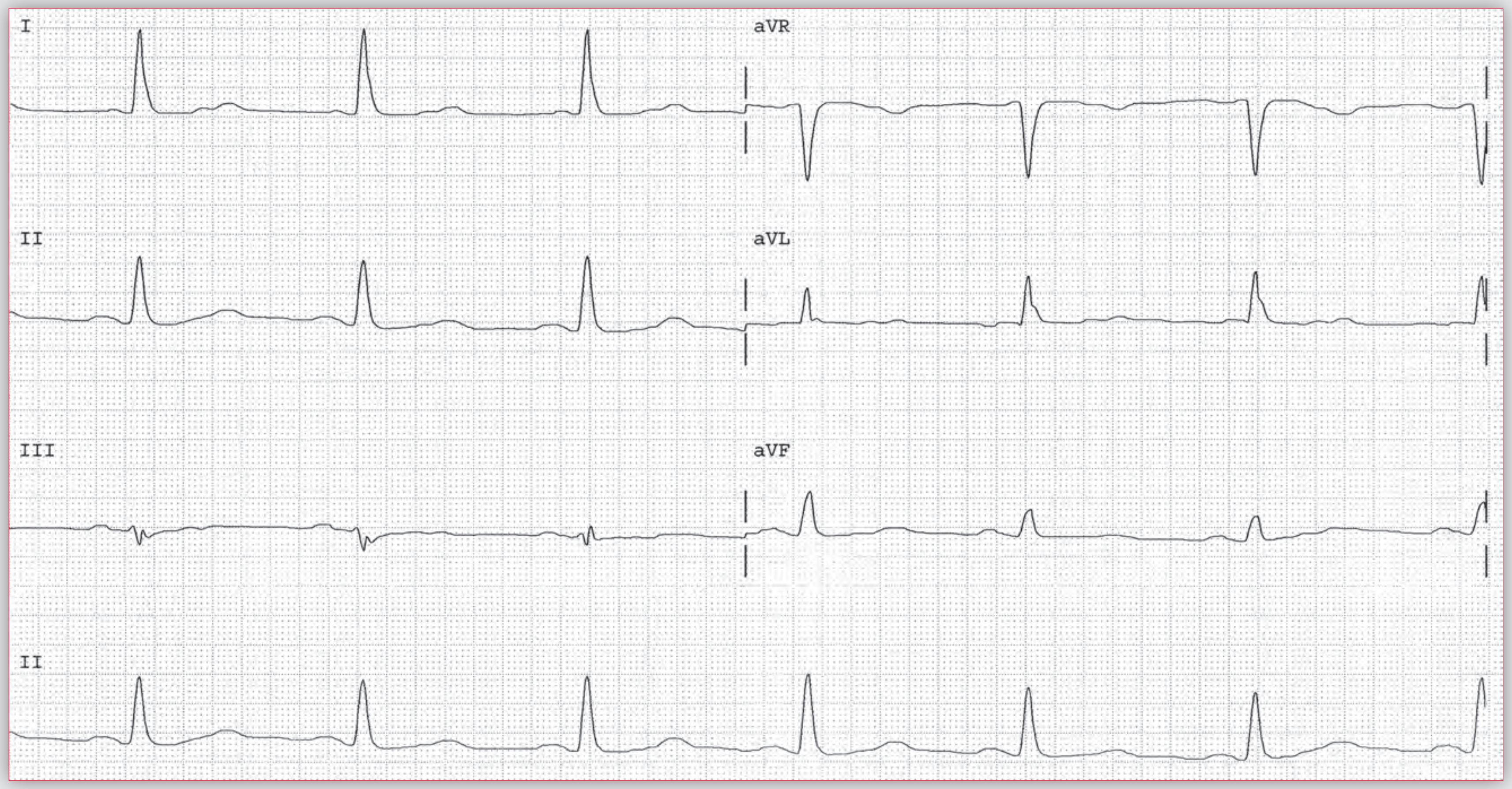
The QRS complex duration is normal (0.08 sec) and there is a rightward axis between +90° and +180° (QRS negative in lead I and positive in lead aVF). Since the QRS voltage is very low (QRS < 5 mm in each limb lead and < 10 mm in each precordial lead), it is hard to establish if there are any abnormalities in the QRS complex morphology to account for the right axis. This is likely a left posterior fascicular block. It is noted that the ECG was recorded at normal standardization (→) (1 mv = 10 mm or boxes), hence there is true low voltage present.

Low voltage reflects a reduction in the amount of electrical current reaching the surface of the body to be recorded by the electrodes, which may be seen with extensive myocardial disease or infiltration (*eg*, amyloid), with a pericardial effusion, with a thick pericardium, in the setting of severe lung disease (COPD), or obesity. The history of an acute onset of acute cardiogenic shock occurring after a respiratory infection suggests that the diagnosis is a fulminant myocarditis. The low voltage is the result of diffuse inflammation and edema of the myocardium that results in a reduction in the magnitude of electrical current. ■

Core Case 52

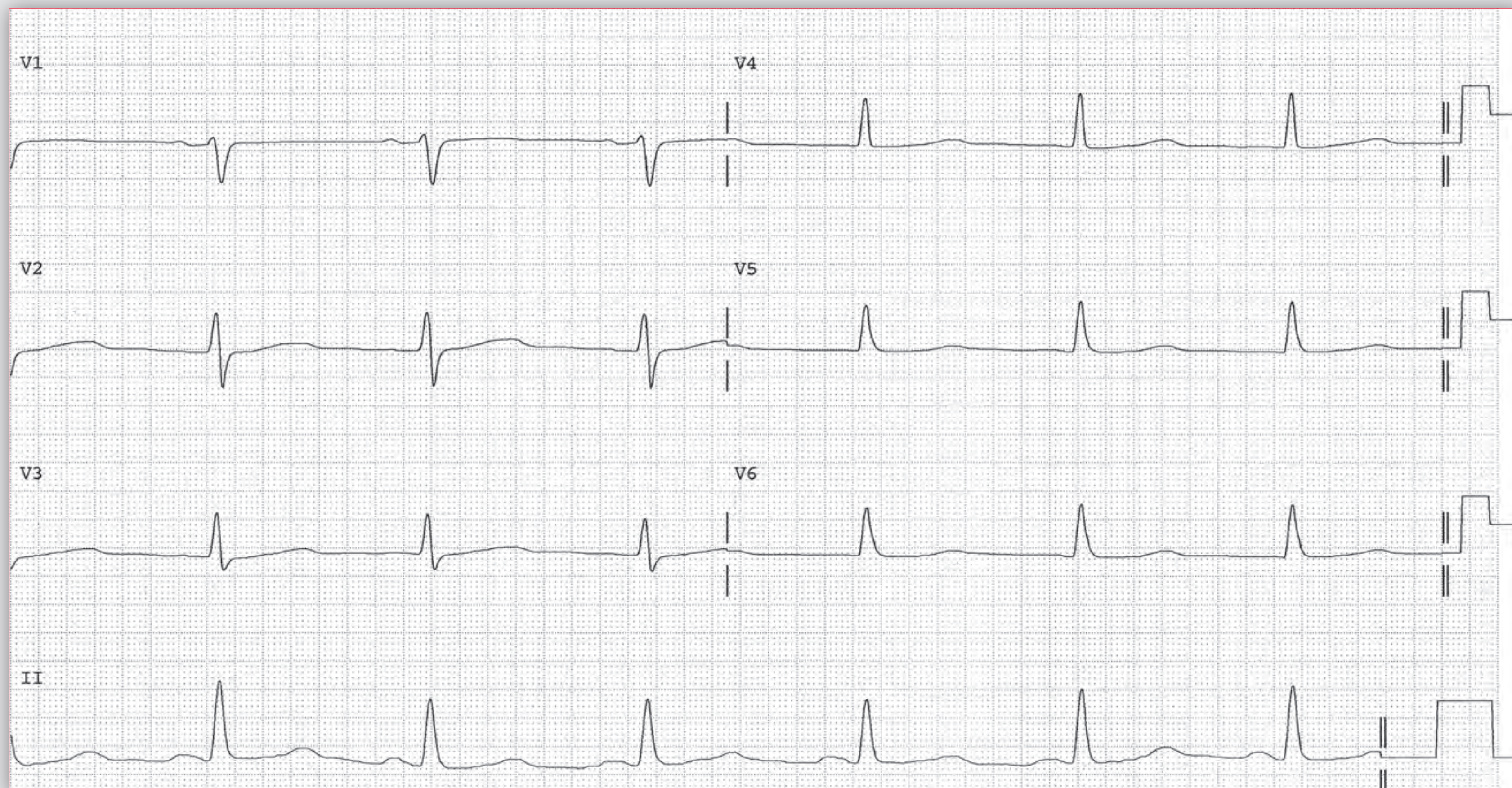
A 42-year-old man is seen for a routine annual physical exam. He is asymptomatic and has no medical diagnoses. He has a family history notable for multiple family members with idiopathic bradycardia requiring pacemaker implantation and some with dilated cardiomyopathy. He has not had any symptoms consistent with these diagnoses and annual screening ECGs and echocardiograms have been normal. ECGs (ECG 52A and 52B) are obtained during the visit. The primary doctor is quite concerned with the tracings obtained and

ECG 52A



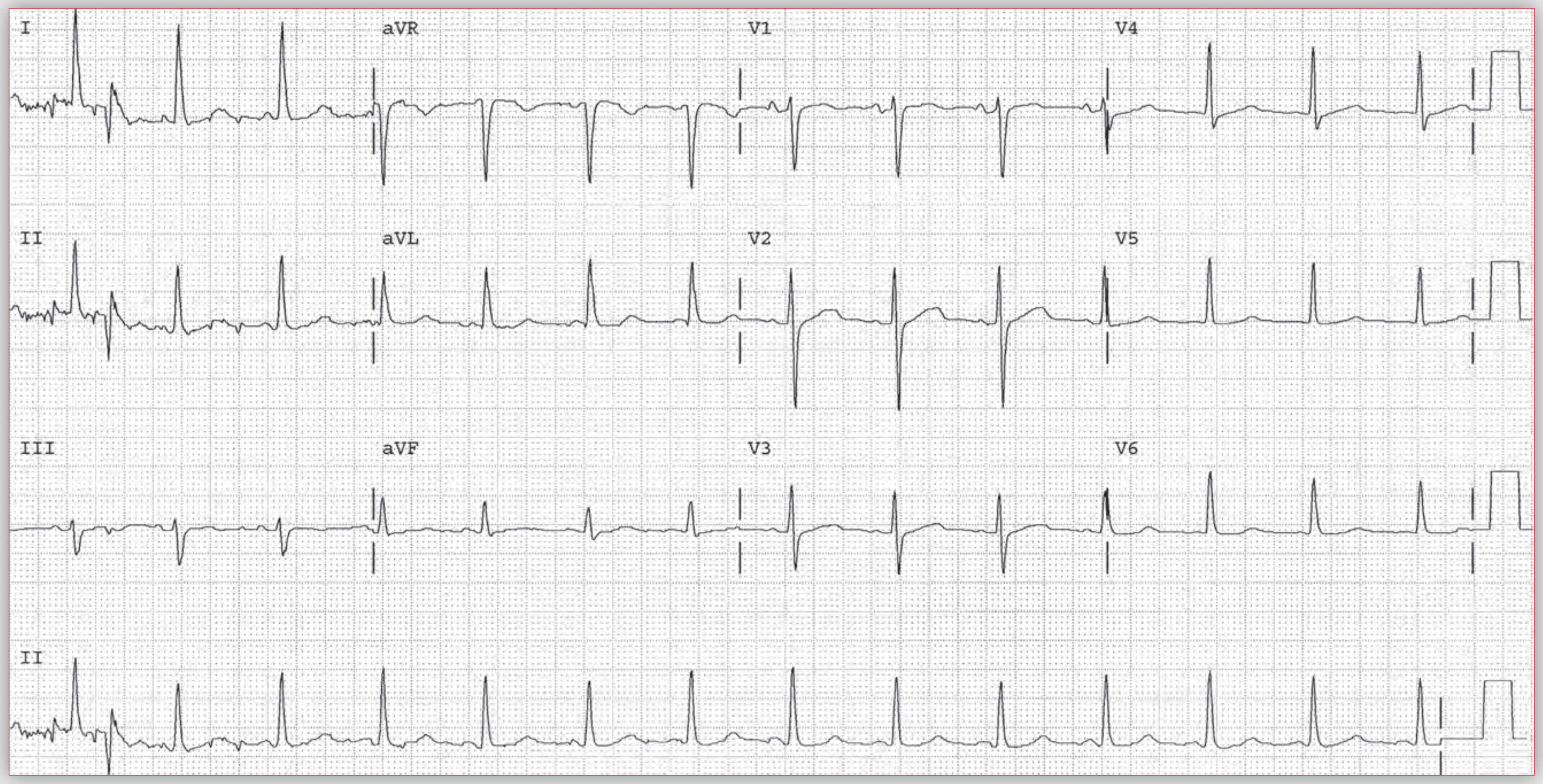
requests a cardiologist in his group to assist with its interpretation, given the patient's ECG 1 year ago was entirely normal. The cardiologist reviews the ECG and requests the primary doctor repeat the tracing rather than proceed with further testing. The patient returns and a follow-up tracing is obtained (ECG 52C).

ECG 52B



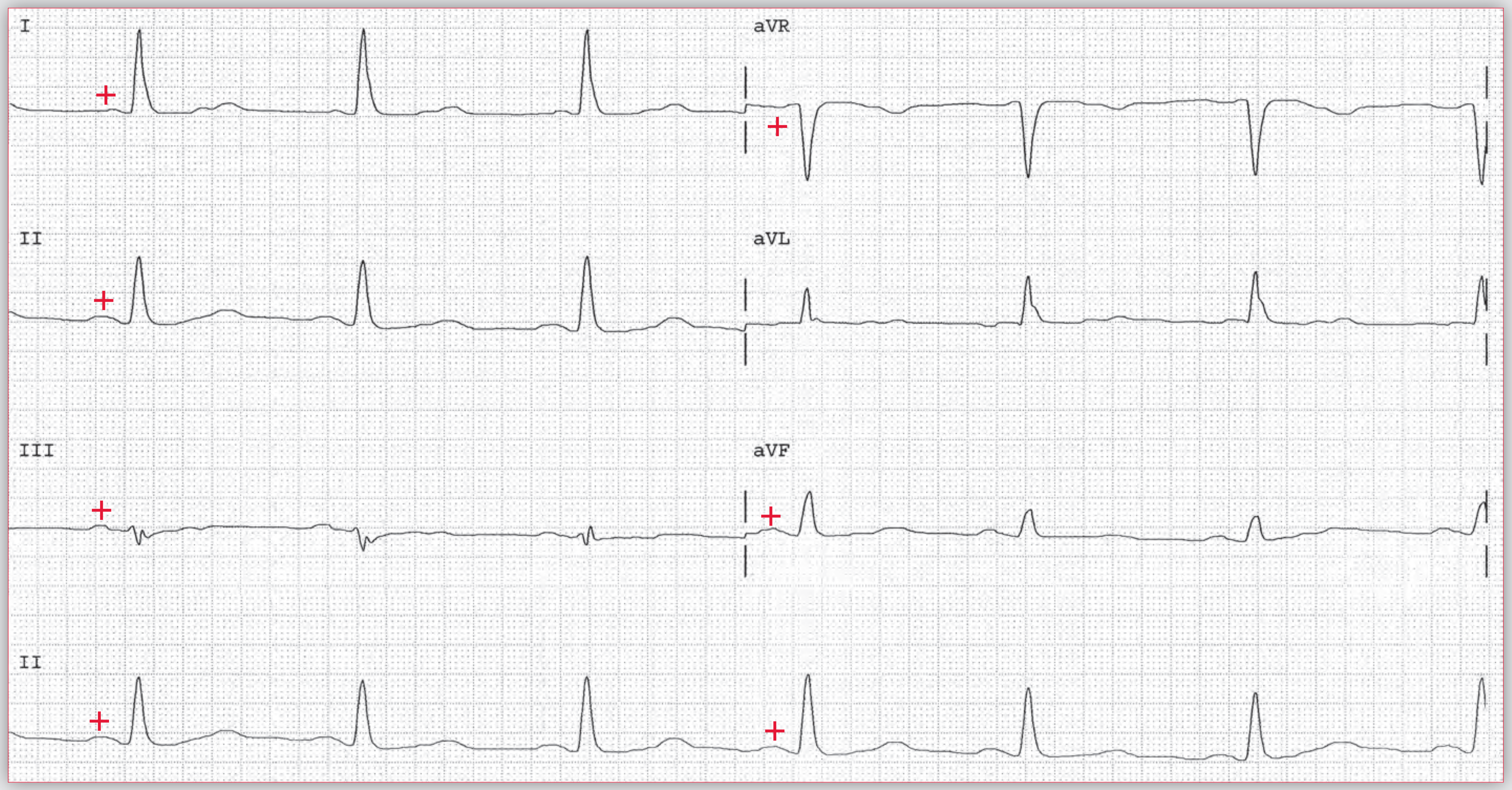
Core Case 52

ECG 52C



What abnormalities as seen in tracing 52A and 52B?

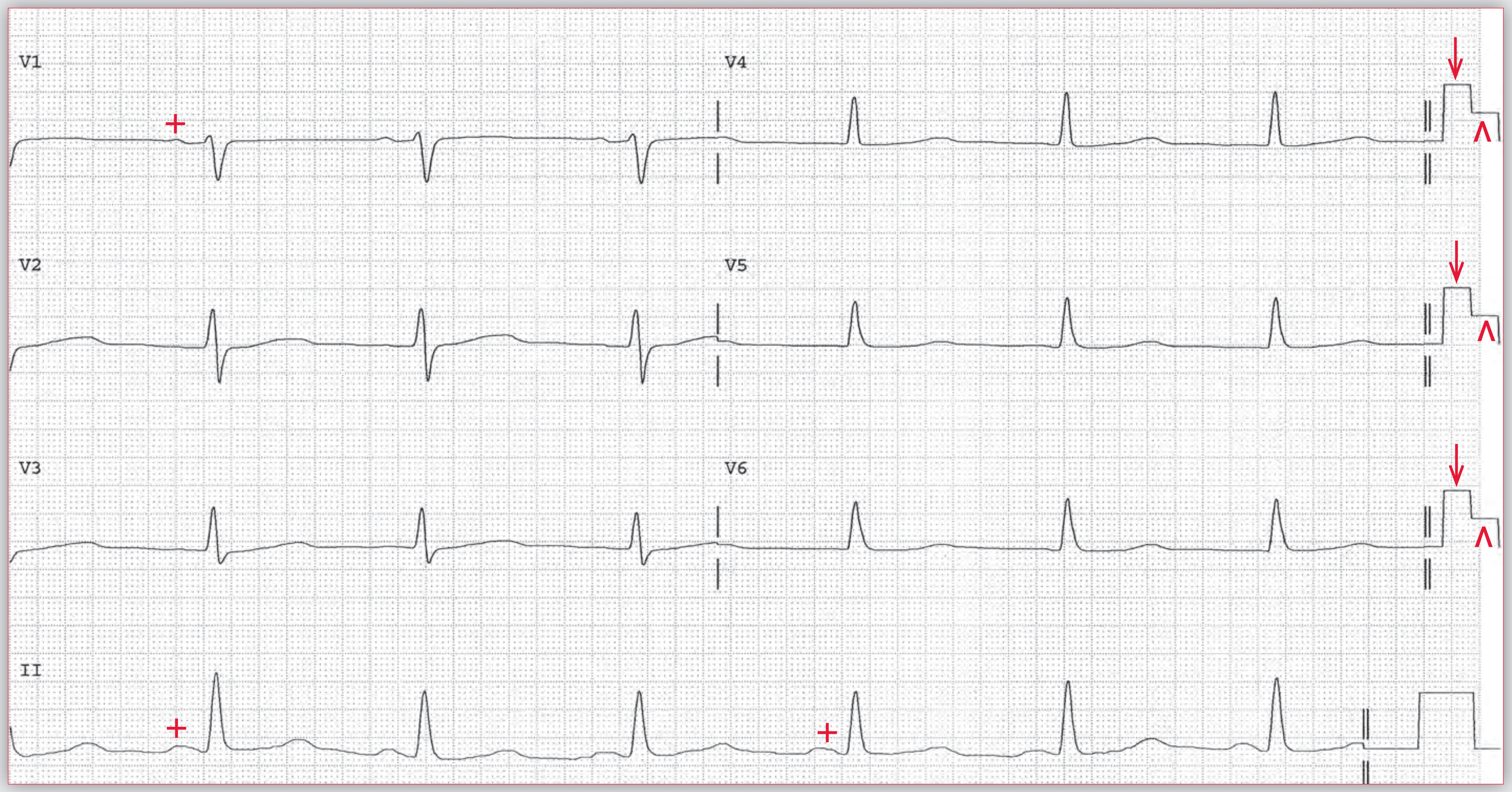
What explains the difference between tracing 52C and the proceeding two?



ECG 52A (and B) Analysis: ECG recorded at 50 mm/sec or double speed

ECG 52A and **52B** shows there is a regular rhythm at a rate of 38 bpm. There is a P wave (+) in front of each QRS complex with a stable PR interval (0.32 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a sinus rhythm. The QRS complex duration is prolonged (0.18 sec) and the QT interval is also prolonged (0.80 sec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). Thus there is an apparent bradycardia as well as prolonged PR, QRS, and QT intervals. Also

continues



ECG 52B (and A) Analysis: ECG recorded at 50 mm/sec or double speed, precordial leads recorded at half standard

noted is that there are only six leads (limb leads) seen. **ECG 52B** shows the six precordial leads and all the intervals are prolonged and are the same as the intervals on ECG 52A. Hence this ECG was recorded at double speed, *ie*, 50 mm/sec, rather than normal speed of 25 mm/sec. Therefore, the actual rate is twice what is measured (*ie*, 76 bpm) and the intervals are half of what is measured, *ie*, the PR is 0.16 sec, QRS is 0.09 sec, and the QT is 0.40 sec.

Also noted, based on the standardization indicated on ECG 52B, the limb leads were recorded at normal standard (↓) (1 mV = 10 mm or boxes) and the precordial leads were recorded at half standard (1 mV = 5 mm or boxes). The normal QRS voltage in the precordial leads is twice what is measured.

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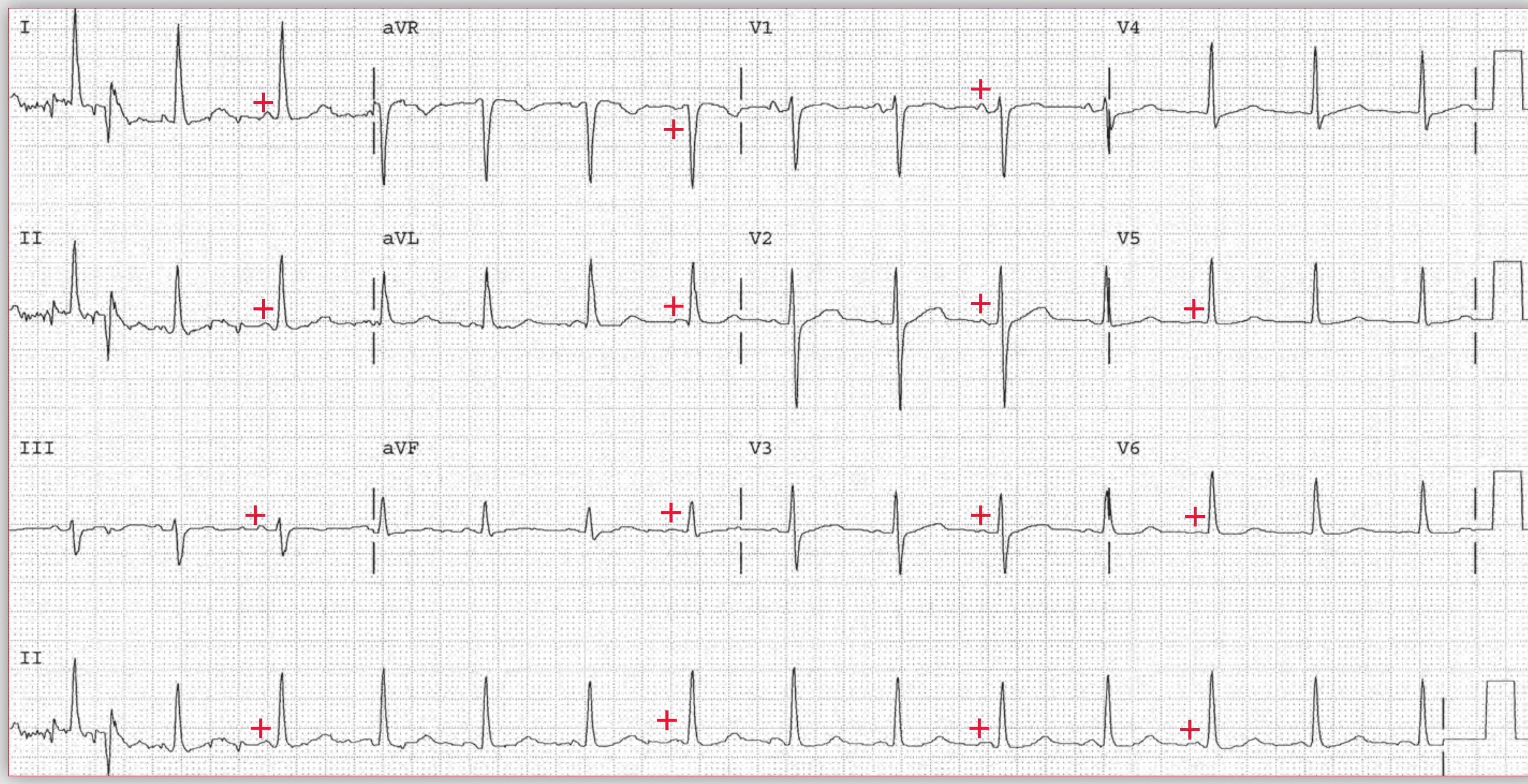


ECG 52C Analysis: ECG recorded at standard paper speed of 25 mm/sec.
Normal sinus rhythm, normal ECG

ECG 52C is from the same patient as ECGs 52A and 52B, but was recorded at normal speed, *ie*, 25 mm/sec, and all the leads were recorded using normal standardization (*ie*, 1 mV = 10 mm or little boxes). The rate is regular at a rate of 86 bpm. There is a P wave (**+**) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/430 msec).

Although many ECG machines will indicate the paper speed used to record the ECG, if this is not indicated, the fact that there are only six leads per sheet of paper and the finding of a very slow heart rate and very long PR, QRS, and QT intervals are important indicators that the paper speed was twice normal. ■

Notes

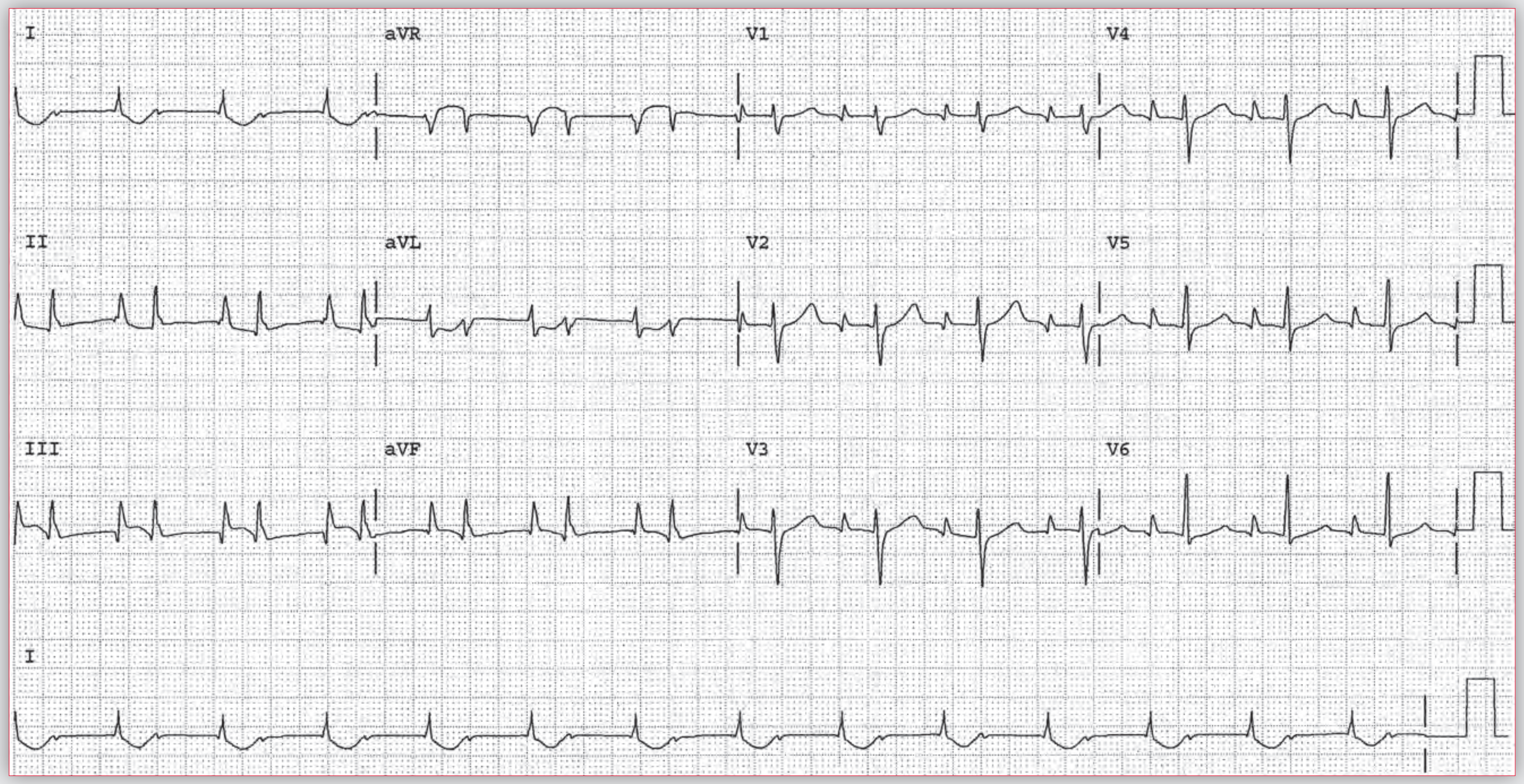
Core Case 53

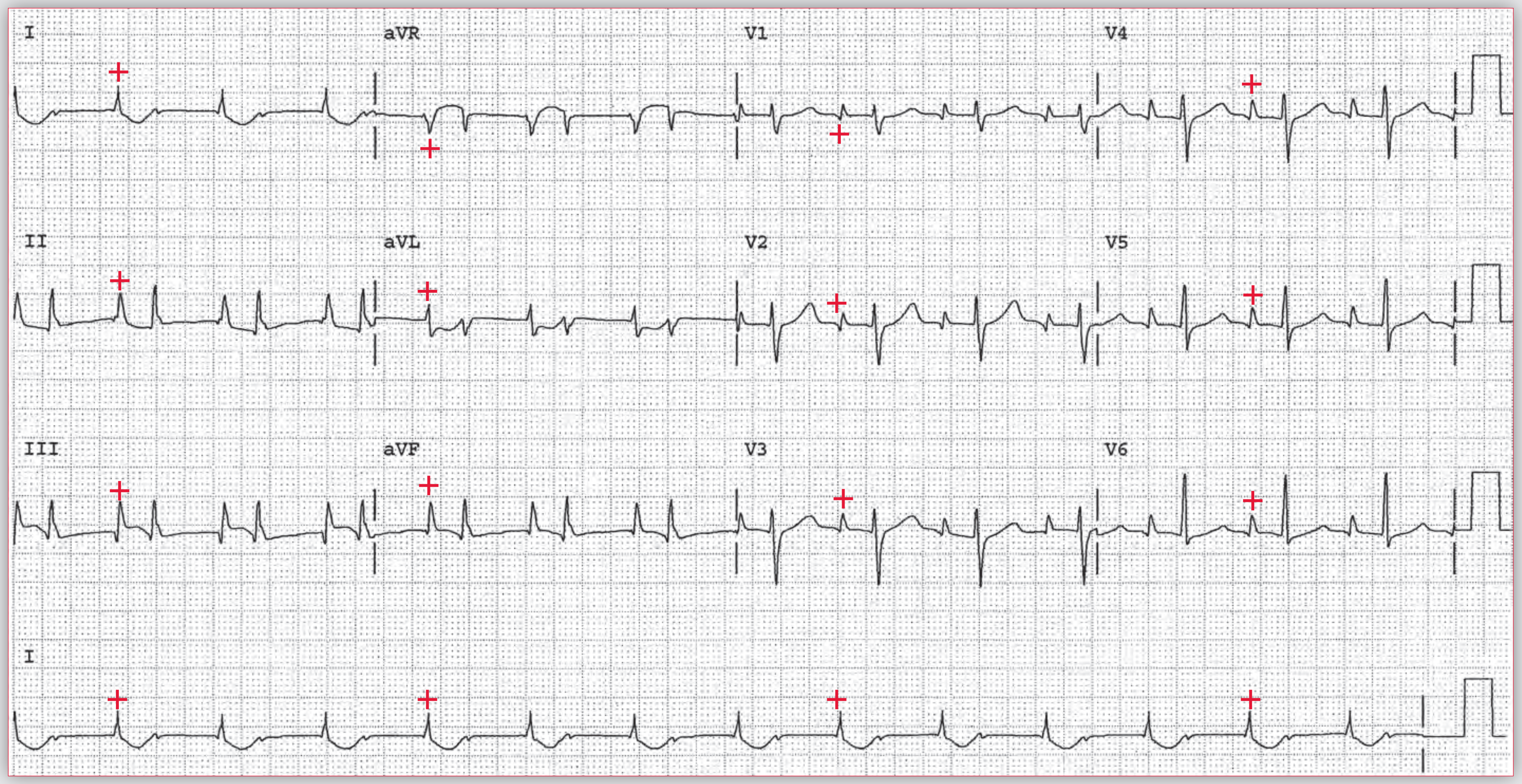
A 69-year-old man is admitted to the hospital because of symptoms of an acute coronary syndrome. He is brought to the catheterization laboratory, where coronary angiography demonstrates a 90% left main lesion and three-vessel disease. As a result the recommendation is for coronary artery bypass surgery. He undergoes three-vessel coronary bypass surgery that is uncomplicated.

Upon arrival to the surgical intensive care unit an ECG is obtained.

Is the ECG normal?

How is the ECG recorded?





ECG 53 Analysis: Sinus rhythm, first-degree AV block, recording from epicardial atrial lead

There is a regular rhythm at a rate of 84 bpm. There is evidence of atrial activity (+) before each QRS complex. There is a stable PR interval (0.24 sec). However, the atrial activity has a very abnormal morphology, with a markedly tall amplitude in some leads (II, III, aVF) being even more prominent than the QRS complex. In some leads the atrial activity is narrow and "spiked" (leads I, aVR, aVL, and V1–V3). This atrial waveform is not the result of a normal ECG recording (*ie*, is not a surface ECG P wave), but rather reflects recording directly from an epicardial atrial lead attached to the right atrium, as is often the case in the patient after cardiac surgery. It is similar to intra-atrial electrogram recording. Although this might be considered atrial pacing, pacing spikes are not seen, and the atrial waveform has a normal duration, unlike the absence of any width or duration of a pacemaker stimulus.

The QRS complexes are normal, although the voltage is very low in the limb leads (QRS < 5 mm in each lead). The QRS complex duration is 0.08 sec and there is normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/430 msec).

Epicardial atrial leads are frequently placed after cardiac surgery, and they are used for recording atrial activity if an atrial arrhythmia should occur. This is especially important to establish the etiology of the arrhythmia particularly if P waves on the surface ECG are not seen. In addition, these leads are often used to attempt overdrive pacing and terminate certain supraventricular arrhythmias. ■

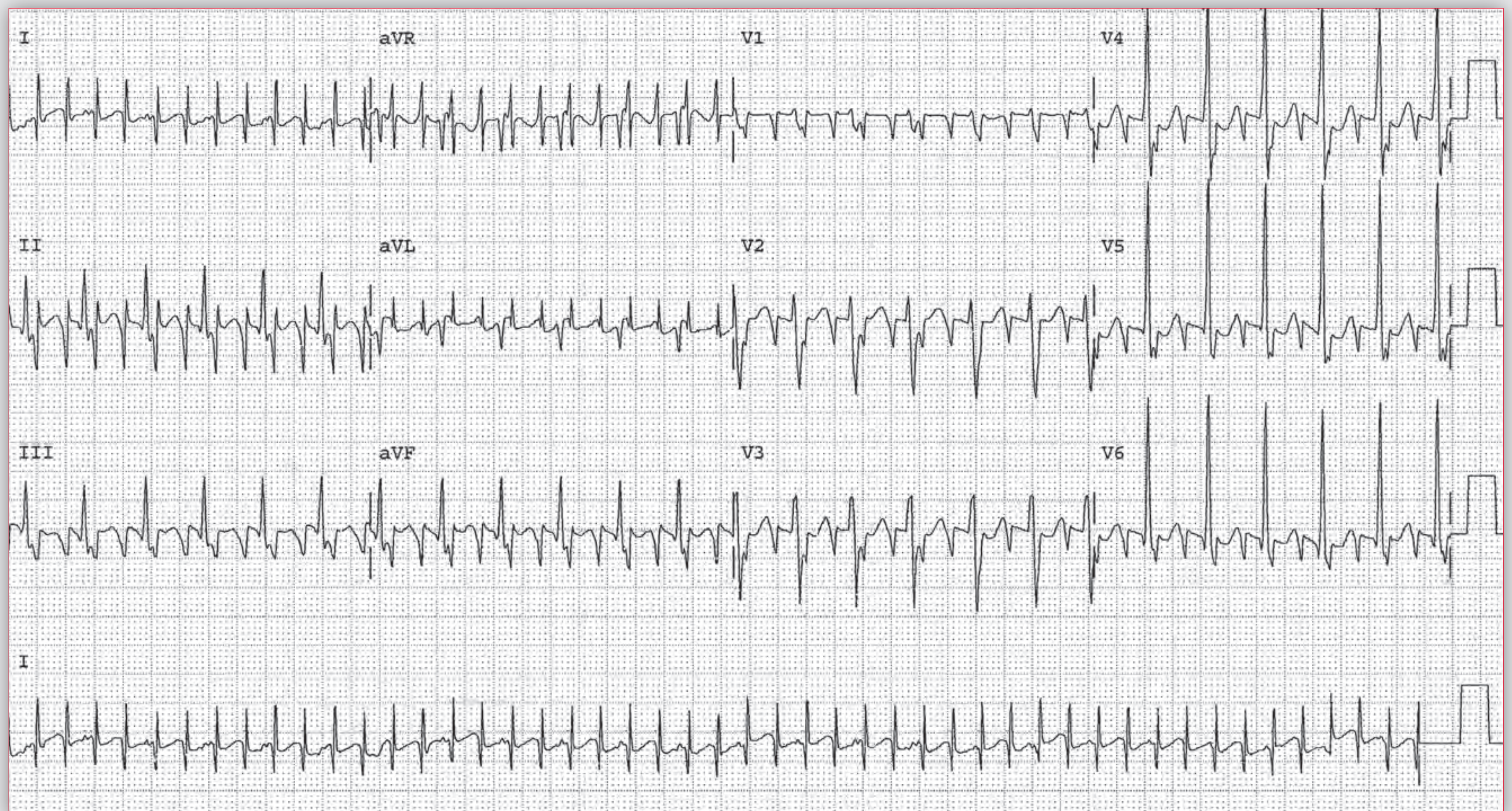
Notes

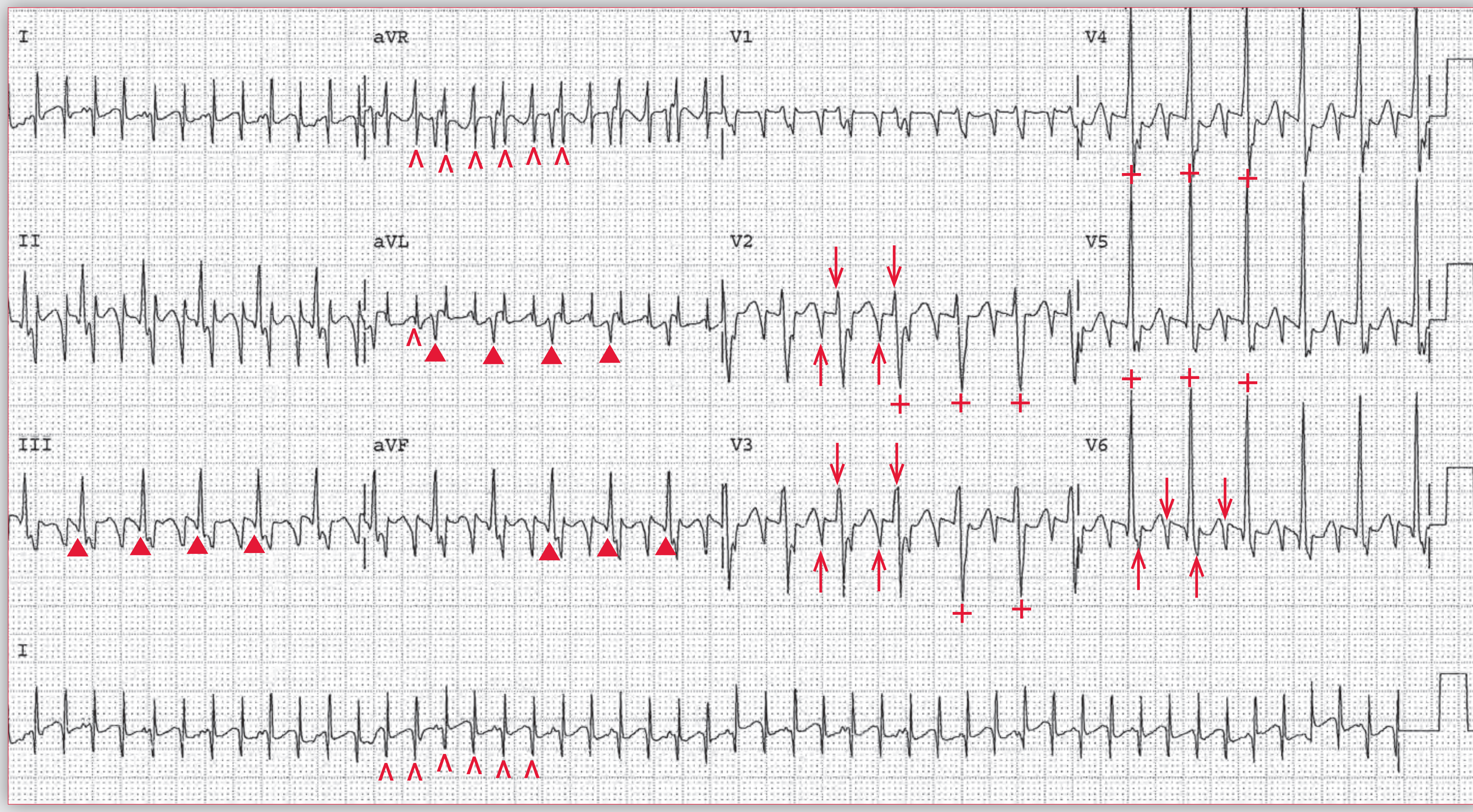
Core Case 54

A 56-year-old patient presenting with shortness of breath and lightheadedness. Evaluation shows that he has critical aortic stenosis, determined to be the result of a bicuspid aortic valve. Valve area is determined to be 0.6 cm^2 and he undergoes an aortic valve replacement. Postoperatively while in the intensive care unit, he develops a supraventricular tachyarrhythmia. An ECG is recorded.

What does the ECG show?

How was the ECG recorded?





ECG 54 Analysis: Atrial flutter, atrial lead recording

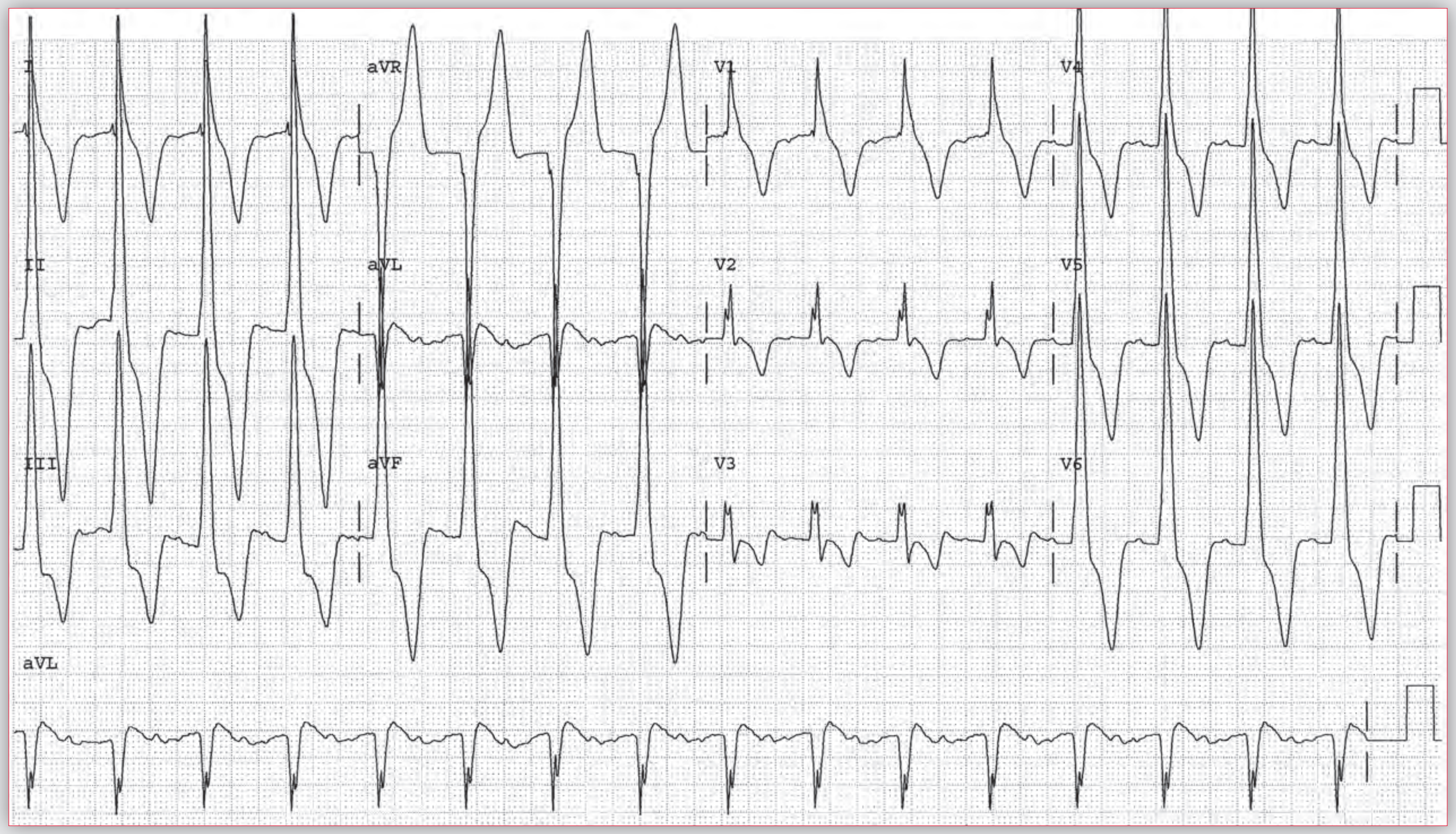
The ECG shows what appears to be a regular rhythm at a rate of 300 bpm (^). In addition, there appears to be electrical or QRS alternans (↓, ↑). However, with closer inspection, it can be seen that there are distinct QRS complexes at a rate of 150 bpm (▲). They are best seen in leads aVL and aVF, seen between the more rapid waveforms that are at a rate of 300 bpm. By timing these QRS complexes, QRS complexes can also be seen in leads V2–V6 (+). The more rapid waveforms represent atrial activity, occurring at a rate of 300 bpm. Hence the underlying rhythm is atrial flutter. The flutter waves are prominent and have a spiked morphology (^), resembling atrial electrograms recorded directly from the atrial myocardium. Hence this ECG was recorded using atrial leads. Therefore, this is atrial flutter with 2:1 AV conduction.

It is common for epicardial atrial leads to be placed after cardiac surgery. They are useful for recording the atrial activity during an atrial tachyarrhythmia. They may also be used for overdrive pacing of an atrial tachyarrhythmia. This may be particularly useful for post-operative atrial flutter. Pacing the atrial at rates slightly above the atrial flutter rate may be effective for termination of the atrial flutter. ■

Core Case 55

A 76-year-old man is admitted to the hospital because of a syncopal episode. A systolic ejection murmur is heard on physical examination, and an echocardiogram confirms aortic stenosis with a valve area of 0.6 cm².

ECG 55A

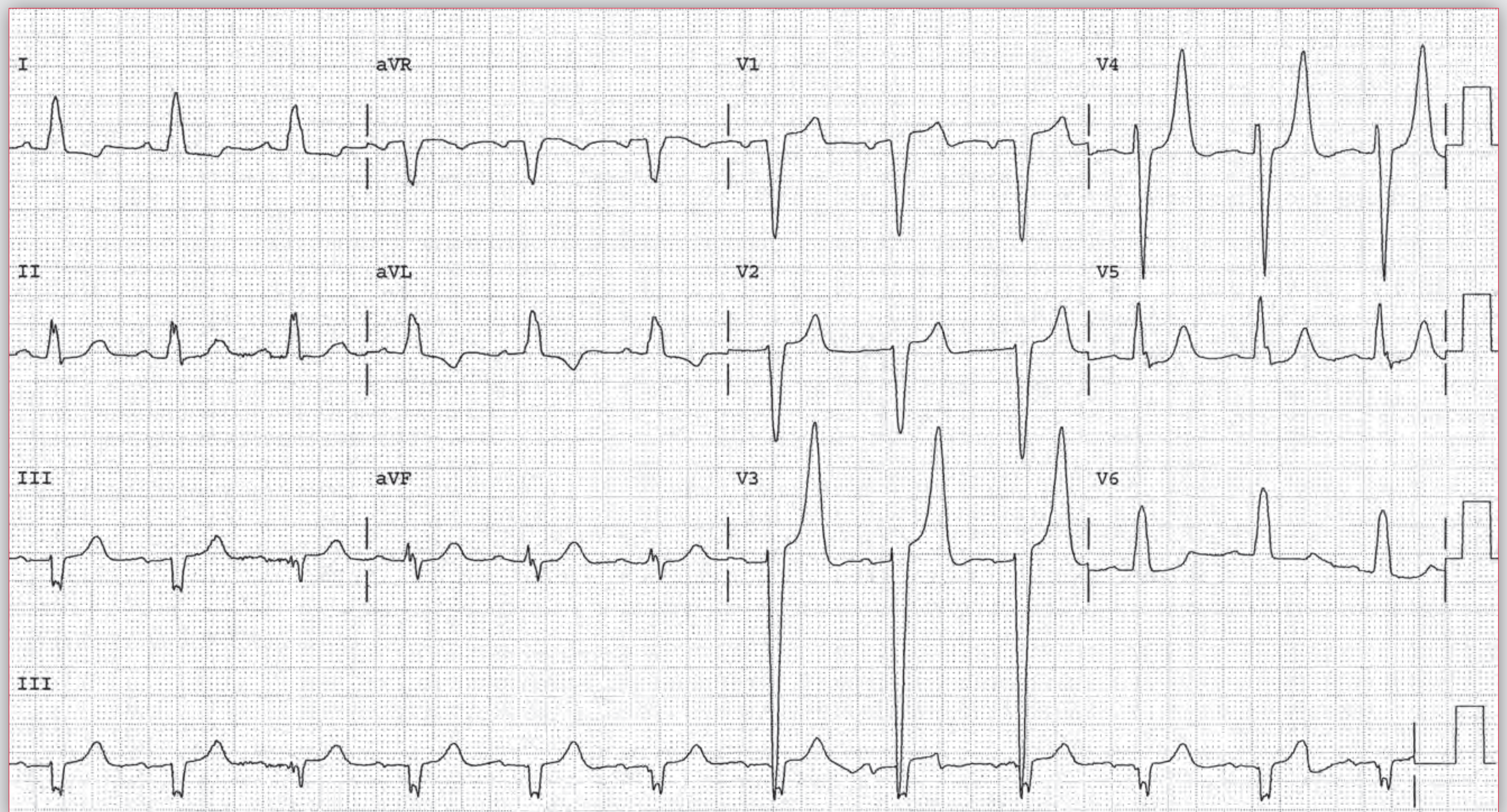


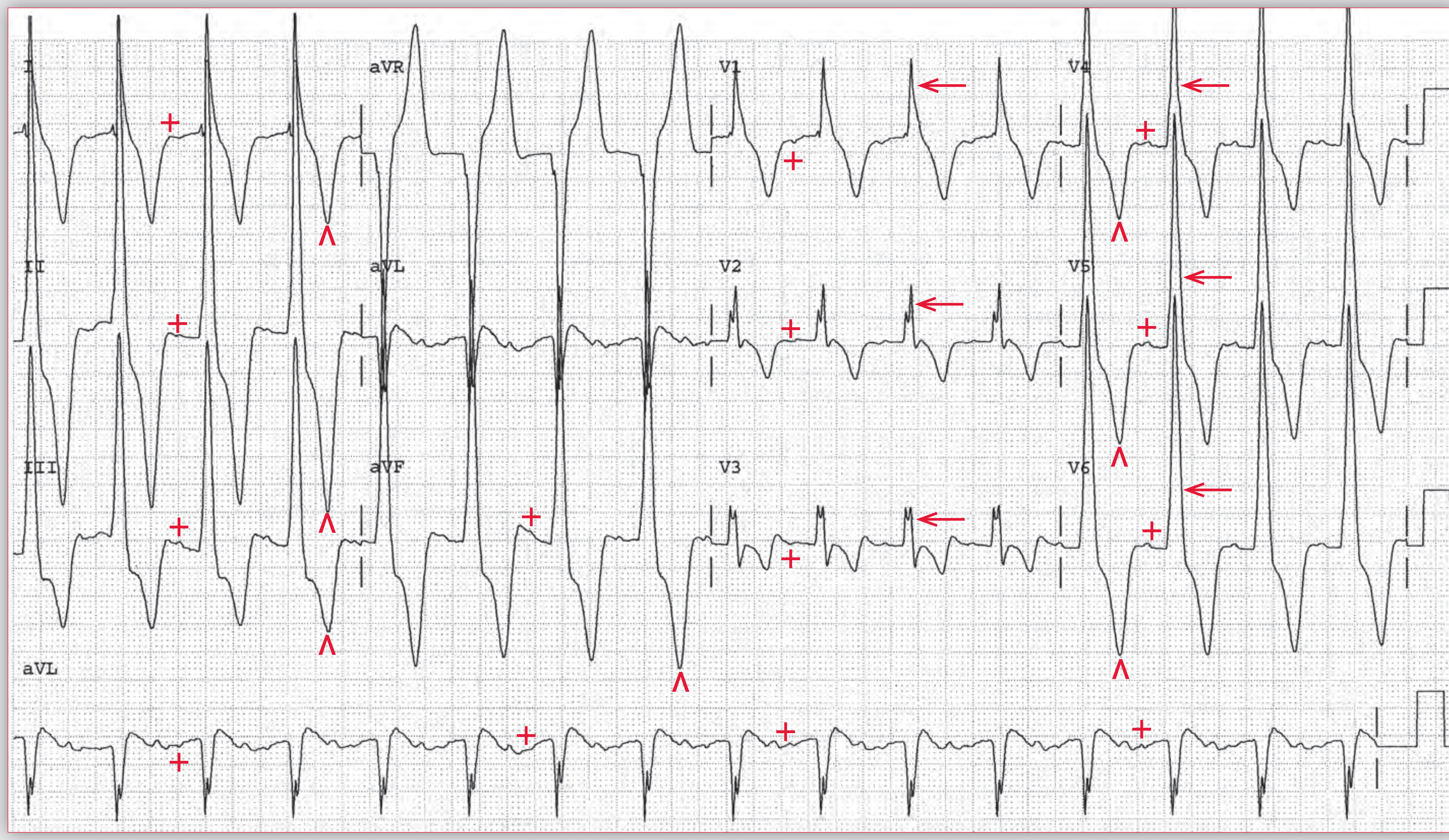
As a result, the patient undergoes an aortic valve replacement. Postoperatively, an ECG is recorded (ECG 55A). The ECG is compared to the baseline ECG (ECG 55B).

Is ECG 55A a normal ECG recording?

How is the ECG recorded?

ECG 55B

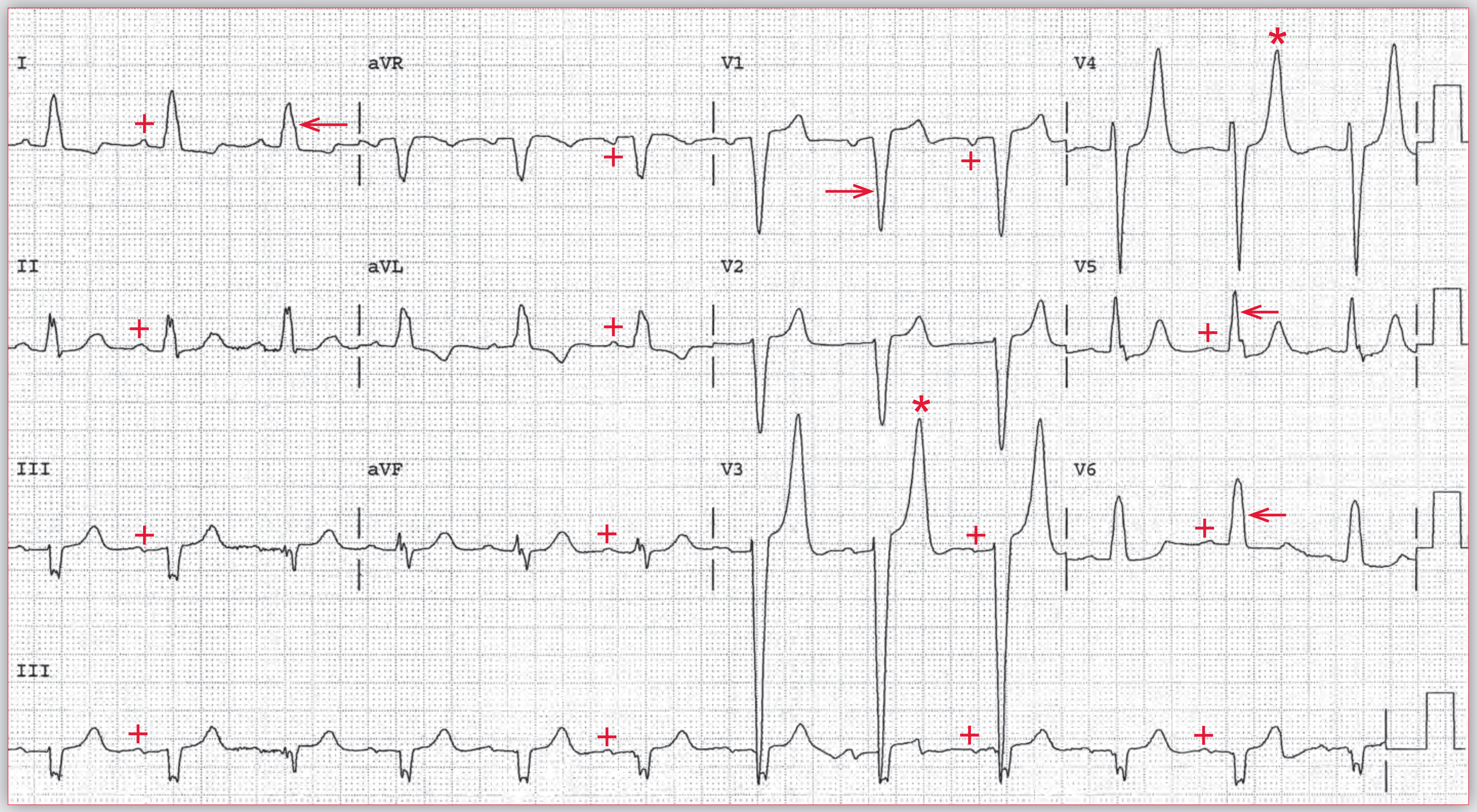




ECG 55A Analysis: Sinus rhythm, first-degree AV block, intraventricular conduction delay (IVCD), left ventricular lead recording

ECG 55A shows there is a regular rhythm at a rate of 94 bpm. There is a P wave (+) before each QRS complex (seen best in leads V4–V6 and leads II and aVF). The PR interval is constant (0.20 sec). The P waves are positive in leads II, aVF, and V4–V6 and hence there is a normal sinus rhythm. The QRS complex duration is prolonged (0.16 sec), but the QRS morphology is not typical of either a right or left bundle branch block. Indeed the QRS morphology is very abnormal. The peak of the QRS complex is "spiked," and there is positive QRS concordance (←) across the precordium (tall R waves V1–V6), which is usually seen whenever there is direct ventricular activation, bypassing the normal His-Purkinje system (*ie*, paced complex, ventricular complexes or in Wolff-Parkinson-White [WPW] pattern). In addition, there is marked ST depression and diffuse, deep T-wave inversions (^). However, there are P waves present before each QRS complex with a fixed relationship or PR interval, meaning that this is a sinus rhythm with intact AV conduction; it is not a paced or a ventricular rhythm. There are no delta waves to suggest WPW. Therefore, the QRS complexes are the result of direct ventricular recording using a temporary pacemaker wire or epicardial lead on the surface of the right ventricle. The spiked look of the QRS complexes resembles a ventricular electrogram. Epicardial leads are routinely placed after cardiac surgery as a precaution in case there is a need for pacing after surgery. Direct epicardial ventricular recording is occasionally performed after cardiac surgery to test lead integrity.

continues



ECG 55B Analysis: Sinus rhythm, first-degree AV block, left bundle branch block

ECG 55B is a standard ECG recorded from the same patient as ECG 55A. There is a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex with a fixed PR interval (0.20 sec). The P wave morphology is normal, and it is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a normal sinus rhythm.

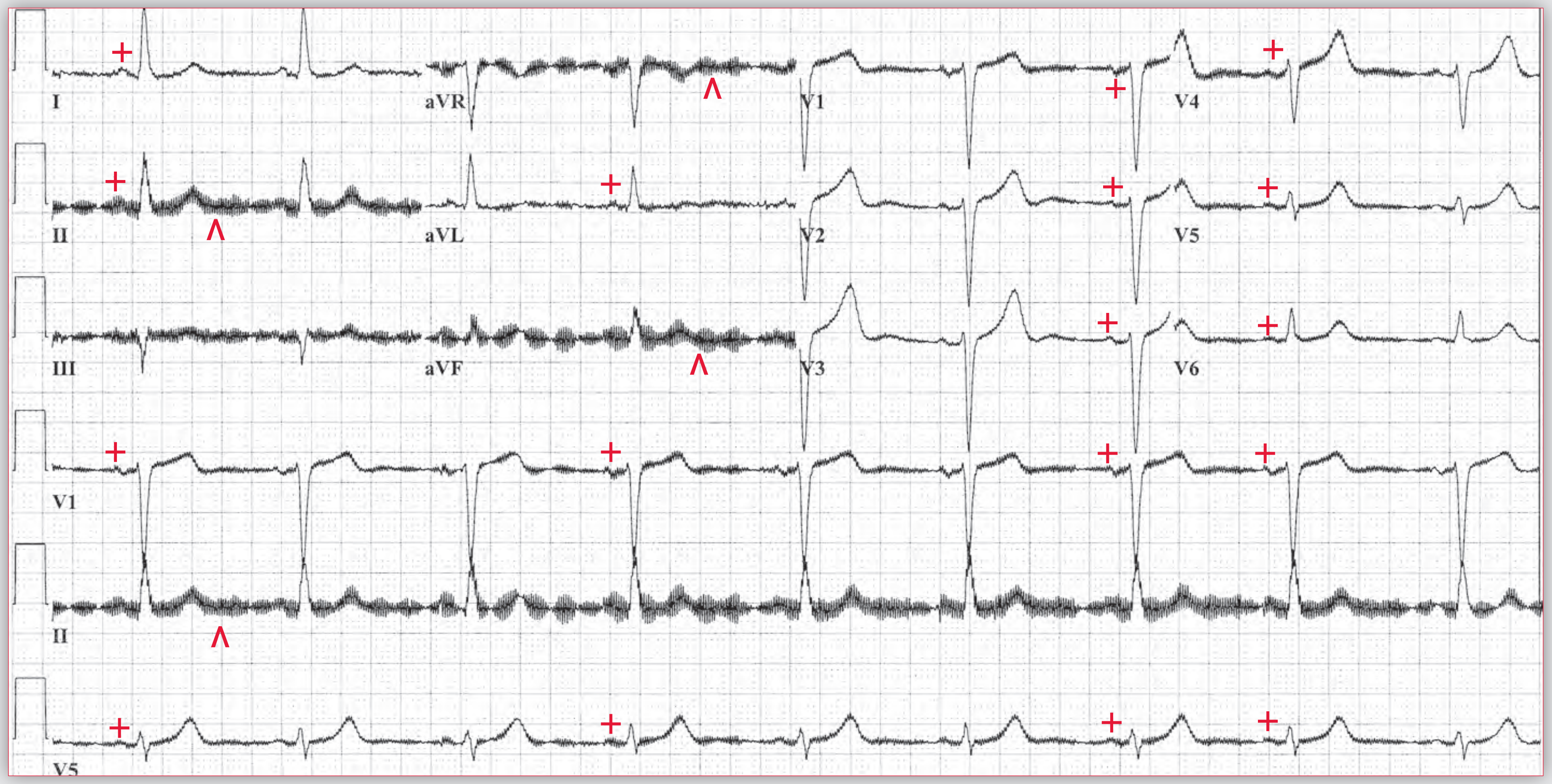
The QRS complex duration is prolonged (0.16 sec) and the morphology is typical for a left bundle branch block with a broad R wave in leads I and V5–V6 (←), and a deep QS complex in lead V1 (→). Although the T waves are very prominent (tall and peaked), especially in leads V3–V4 (*), they are asymmetric and hence normal. The QT/QTc intervals are prolonged (460/500 msec) but are normal when the prolonged QRS complex duration is considered (400/430 msec). The QRS complexes seen with the usual ECG recording are in striking contrast to those seen in ECG 55A that is recorded using a ventricular pacing electrode. ■

Notes

Core Case 56

A 77-year-old woman who has recently undergone an aortic aneurysm repair remains intubated in the SICU because of low O_2 saturations. An ECG is obtained. The cardiologist who is reading ECGs asks for the ECG to be repeated.

What is the abnormality noted that prompted the need for a repeat ECG?



ECG 56 Analysis: Normal sinus rhythm, 60-cycle interference

There is a regular rhythm at a rate of 54 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (↔) (0.16 sec). The P waves are positive in leads I, II, and V4–V6. Hence this is a sinus rhythm. The QRS complex duration is normal (0.10 sec) and there is a normal morphology with a normal axis between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (440/420 msec).

There is very obvious artifact present (^) in leads II, III, aVL, and aVF. This type of artifact is the result of 60-cycle interference, often present in intensive care units where many types of electronic equipment are present. ■

Notes

Core Case 57

A 62-year-old man with prior anterior myocardial infarction (MI) and heart failure presents to the emergency department with palpitations and lightheadedness. His exam is notable for a normal and regular pulse and a normal blood pressure. There is no neck vein distension. His lungs are clear. His prior PMI is laterally displaced and an S3 is noted. A soft, blowing apical murmur is noted. His lower extremities are without edema. He is noted to have a mild tremor. An ECG is obtained which causes the nurse to become alarmed. She stat pages the physician and respiratory therapist.

What abnormalities are depicted?

What findings concerned the nurse?

What is the cause of the abnormalities?

ECG 57 Analysis: Atrial flutter with moderate ventricular response, marked artifact

The ECG shows waveforms that are rapid (rate 280 bpm), regular, wide, and abnormal in morphology. The waveforms resemble a rapid ventricular tachycardia. However, there are obvious narrow QRS complexes (+) noted in lead II, and also atrial flutter waves (^). In addition, close inspection of leads V1, V3, and V4 show distinct QRS complex waveforms (↑) and also waveforms that look like atrial flutter (▼). Since every column is simultaneous, what is seen in lead II is the same as in leads I and III, and what is seen in leads V1, V3, and V4 is the same as the other leads in the same column. Therefore, the rapid and regular waveforms are not ventricular tachycardia, but are artifact, likely the result of the patient's resting tremor. ■

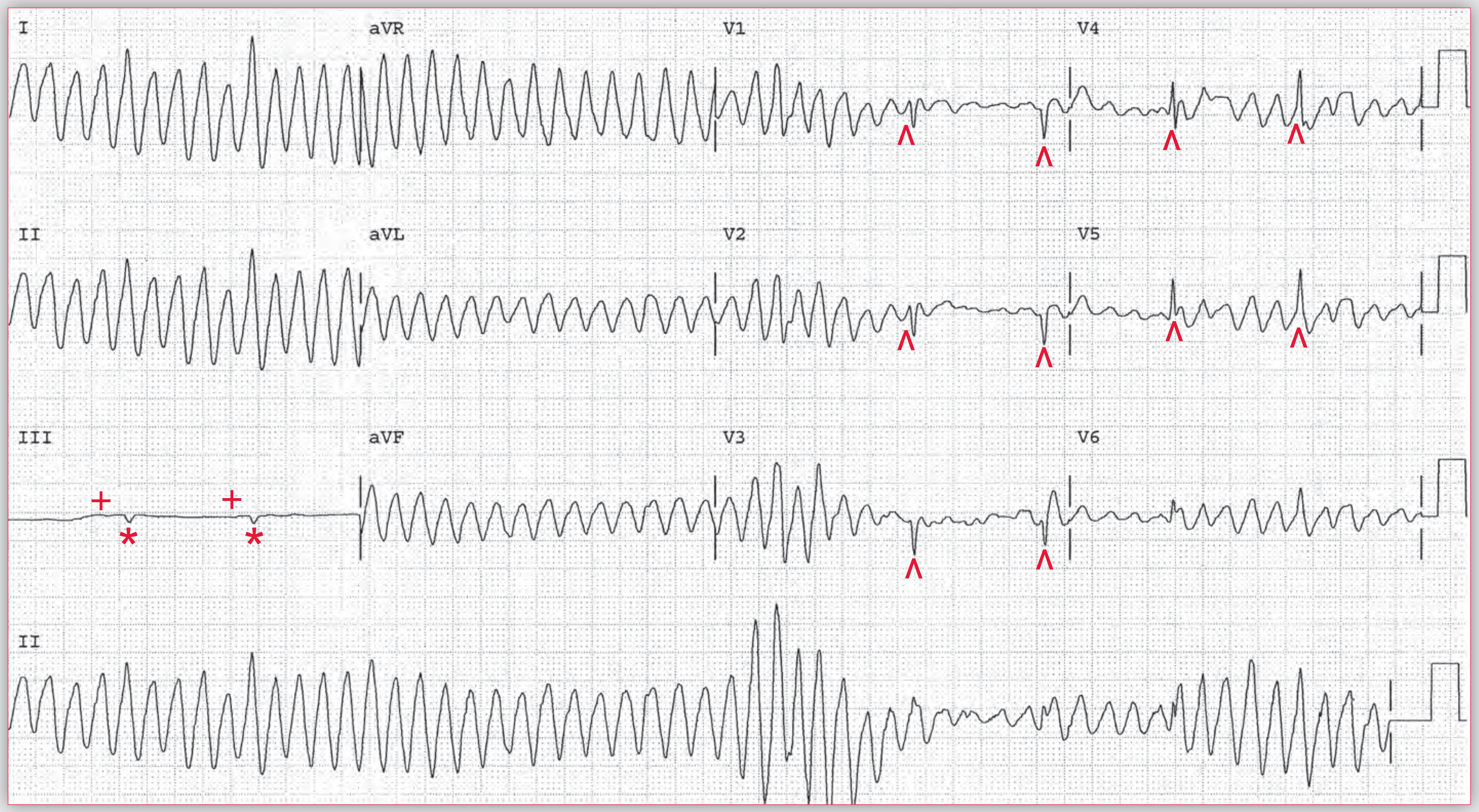
Notes

Core Case 58

A 76-year-old woman with a history of Parkinson's disease presents to the hospital because of substernal chest discomfort. She is admitted to the hospital and placed on telemetry. Several hours later, the nurse observes a rapid and regular wide complex tachycardia, and she calls a code. Upon arrival to the room, the code team notes that the patient is lying comfortably in bed. An ECG is obtained.

What is seen?

Does this explain the abnormality noted on telemetry?



ECG 58 Analysis: Normal sinus rhythm, artifact

The ECG shows waveforms that are rapid, at a regular rate of almost 300 bpm, wide and abnormal. They also appear to have a changing morphology, suggesting polymorphic ventricular tachycardia. Although they resemble QRS complexes seen in ventricular tachycardia, there are distinct narrow QRS complexes (*) preceded by a P wave (+) seen in lead III. These are sinus complexes. As lead III is simultaneous with leads I and II, the abnormal waveforms in these leads are artifact. Indeed, distinct QRS complexes (^) can be seen in leads V1–V6 in between the waveforms that are artifact. Therefore, it is most likely that the monitor demonstrated artifact that was interpreted as an arrhythmia. The artifact is no doubt due to the patient's Parkinson's disease.

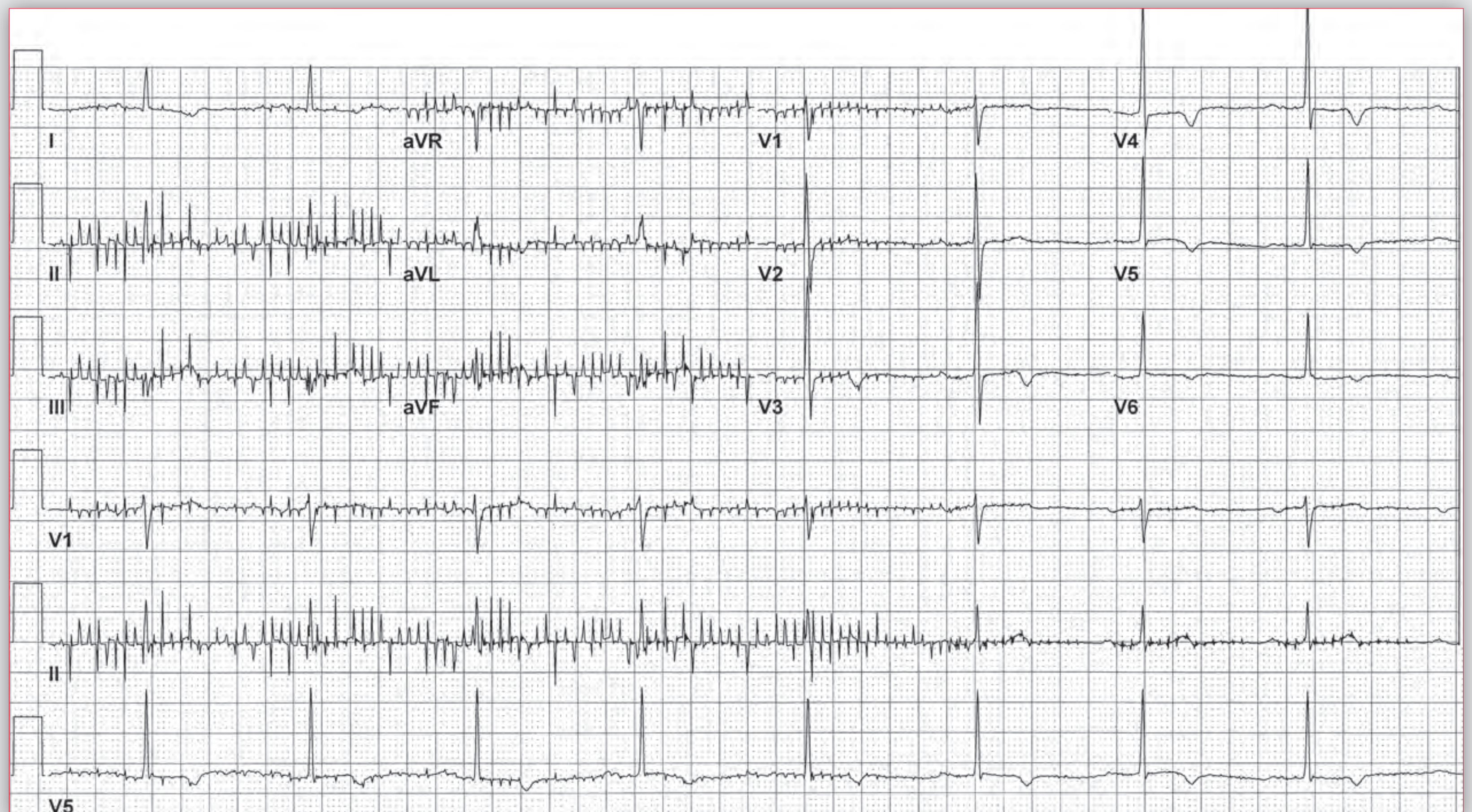
Artifact on a telemetry is very common and may be due to a tremor, patient movement (such as brushing teeth), a loose lead connection, lead fracture, or the patient twiddling with the telemetry leads. ■

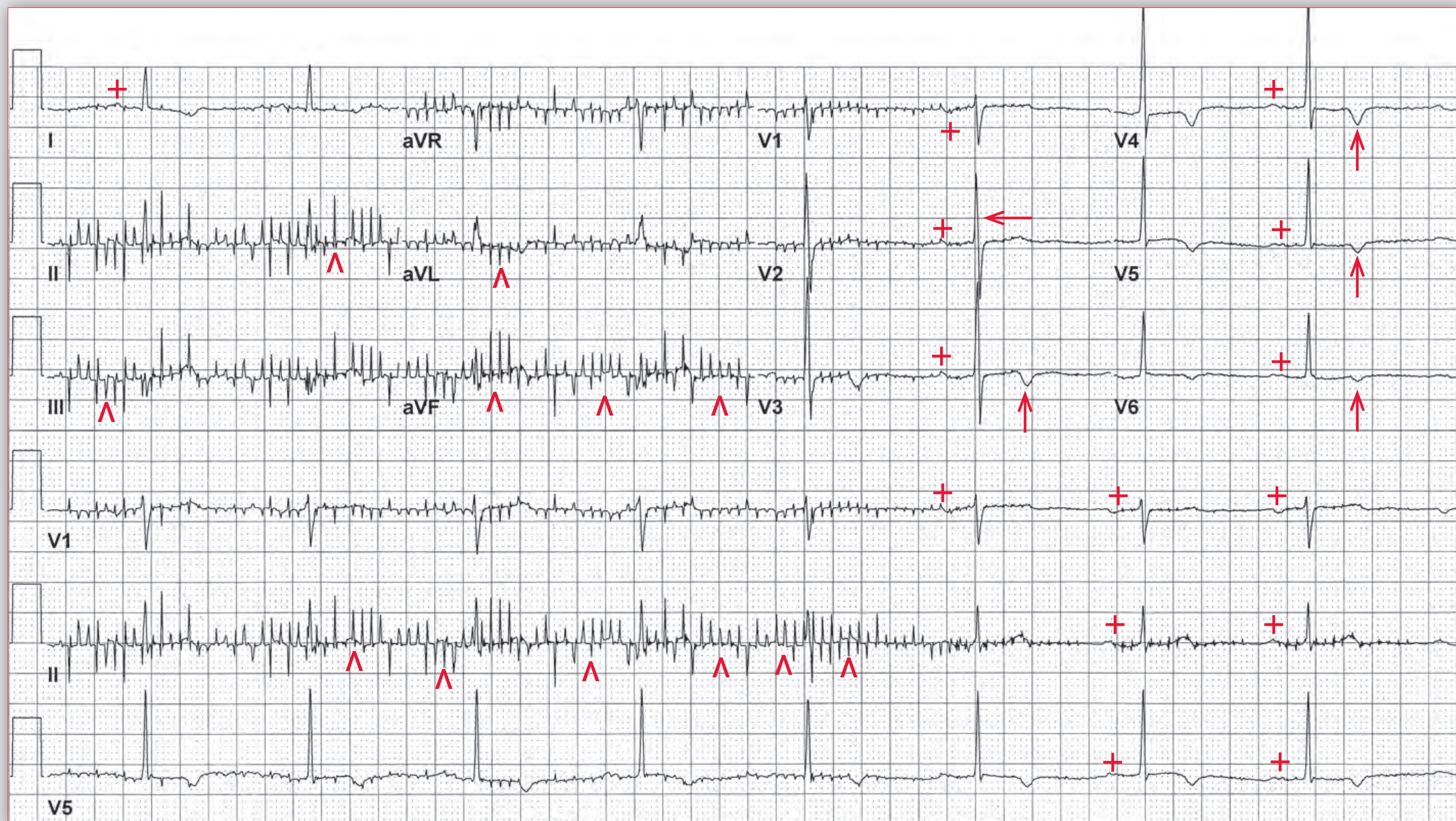
Notes

A 72-year-old female is admitted to the general medical service with a complicated urinary tract infection. Her medical history is notable for symptomatic bradycardia requiring pacemaker implantation, recalcitrant incontinence for which she has a bladder stimulator and osteoporosis. An ECG is obtained when her physical exam documents bradycardia. The medical intern presents the patient's case to her attending and wonders whether the cardiac electrophysiology service should be consulted for a malfunctioning pacemaker.

What abnormalities are noted on the ECG?

Do you agree on the intern's interpretation of the ECG and why?





ECG 59 Analysis: Sinus bradycardia with first-degree AV block, counterclockwise rotation, nonspecific T-wave changes, artifact due to bladder stimulation

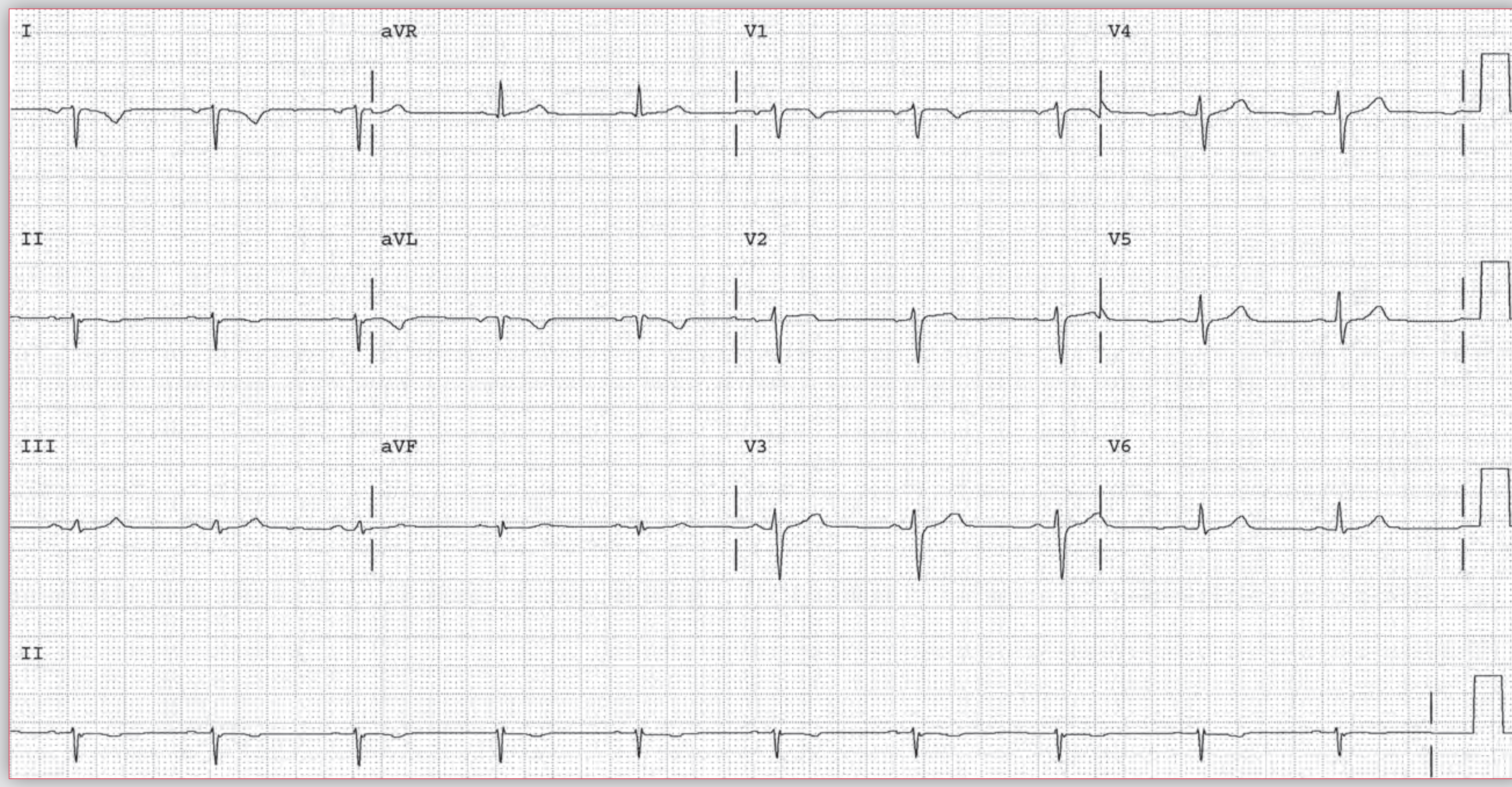
There is a regular rhythm at a rate of 52 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.28 sec). The P waves can be seen primarily in leads I and V4–V6 and are positive, representing a sinus rhythm with a first-degree AV block. QRS complexes are not visible in leads II, III, aVR, aVL, and aVF as a result of artifact (^), which interferes with their identification. However, the QRS complexes are well seen in the precordial leads. They are narrow (0.08 sec) and have a normal morphology. There is a tall R wave in lead V2 (←) due to early transition or counterclockwise rotation of the axis in the horizontal plane. This is established by imagining the heart as if viewed from under the diaphragm. With counterclockwise rotation, left ventricular forces are directed anteriorly, accounting for the tall R wave in V2. The QT/QTc intervals are normal (440/410 msec). There are nonspecific T-wave abnormalities (↑) in V3–V6). Although the artifact looks like pacemaker stimuli, they are actually the result of an external bladder stimulator.

It is not likely that a pacemaker stimulus could occur at this very rapid rate. Moreover, a pacemaker may fail to sense the native P wave or QRS complex or, such as in the case of a lead fracture, may fail to regularly pace the cardiac chambers. Neither scenario would lead to random pacing spikes on a surface ECG. A failure to sense would result in inappropriate but occasional successful pacing. A lead fracture would result in a failure to pace on a regular basis. This patient's pacemaker is likely functioning normally and set to initiate pacing at a heart rate below 52 bpm as no pacemaker stimuli are noted. ■

Core Case 60

A previously healthy 44-year-old man is seen in the emergency department for epigastric discomfort that he had initially attributed to indigestion. However, his physician is concerned when he sees his ECG (ECG 60A). As a result he asks for the ECG to be repeated (ECG 60B).

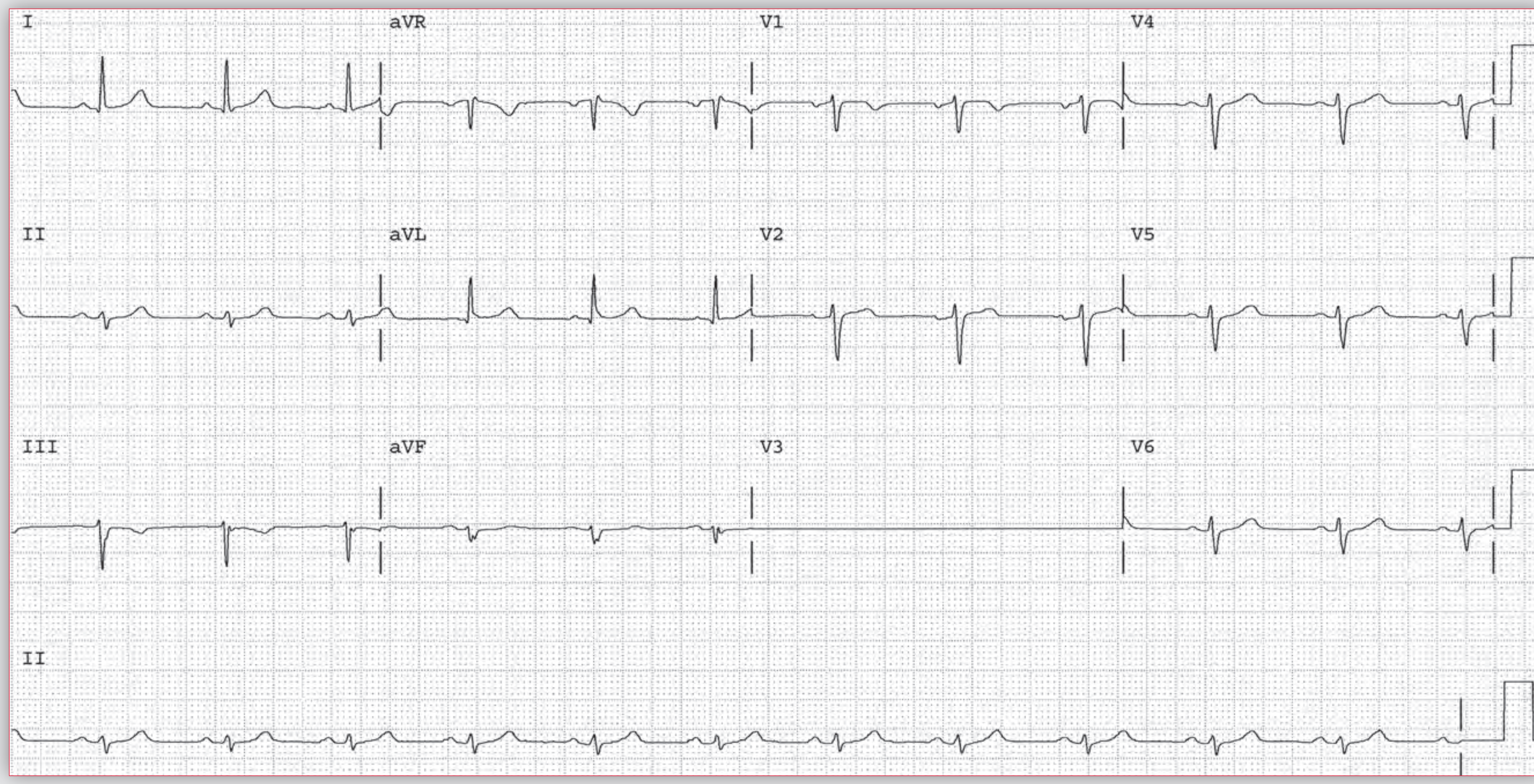
ECG 60A

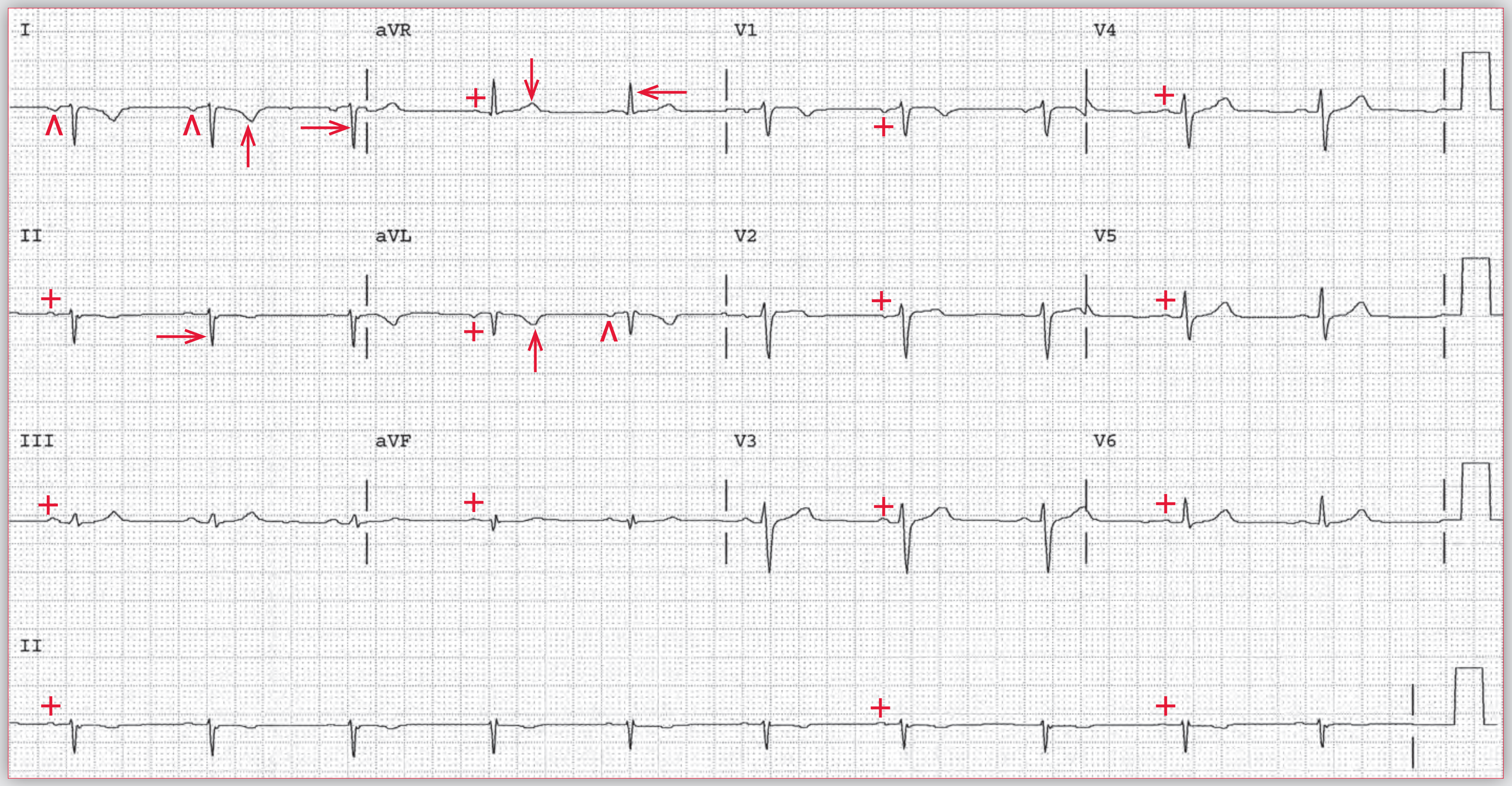


What does the ECG show?

What is the most likely explanation?

ECG 60B





ECG 60A Analysis: Normal sinus rhythm, right-left arm lead switch

ECG 60A shows there is a regular rhythm at a rate of 60 bpm with a P wave (+,^) before each QRS complex and a stable PR interval (0.16 sec). The P wave is negative (^) in leads I and aVL, while it is positive in leads II, aVR, aVF, and V4–V6.

The QRS complex duration is normal (0.08 sec). The axis in the frontal plane is strange (indeterminate) as the QRS complex is negative in leads I and II (→) and biphasic in lead aVF. There is also a tall R wave in lead aVR (←). QRS morphology is normal in the precordial leads. The QT/QTc intervals are normal (400/400 msec). The T waves are negative in leads I and aVL (↑) and are positive in lead aVR (↓). The strange axis and negative QRS, P wave and T waves in leads I and aVL and the positive QRS complex, positive P wave, and positive T wave in lead aVR are changes characteristic of a right-left arm lead switch, such that leads I, aVR, and aVL are upside down. The QT/QTc intervals are normal (400/400 msec).

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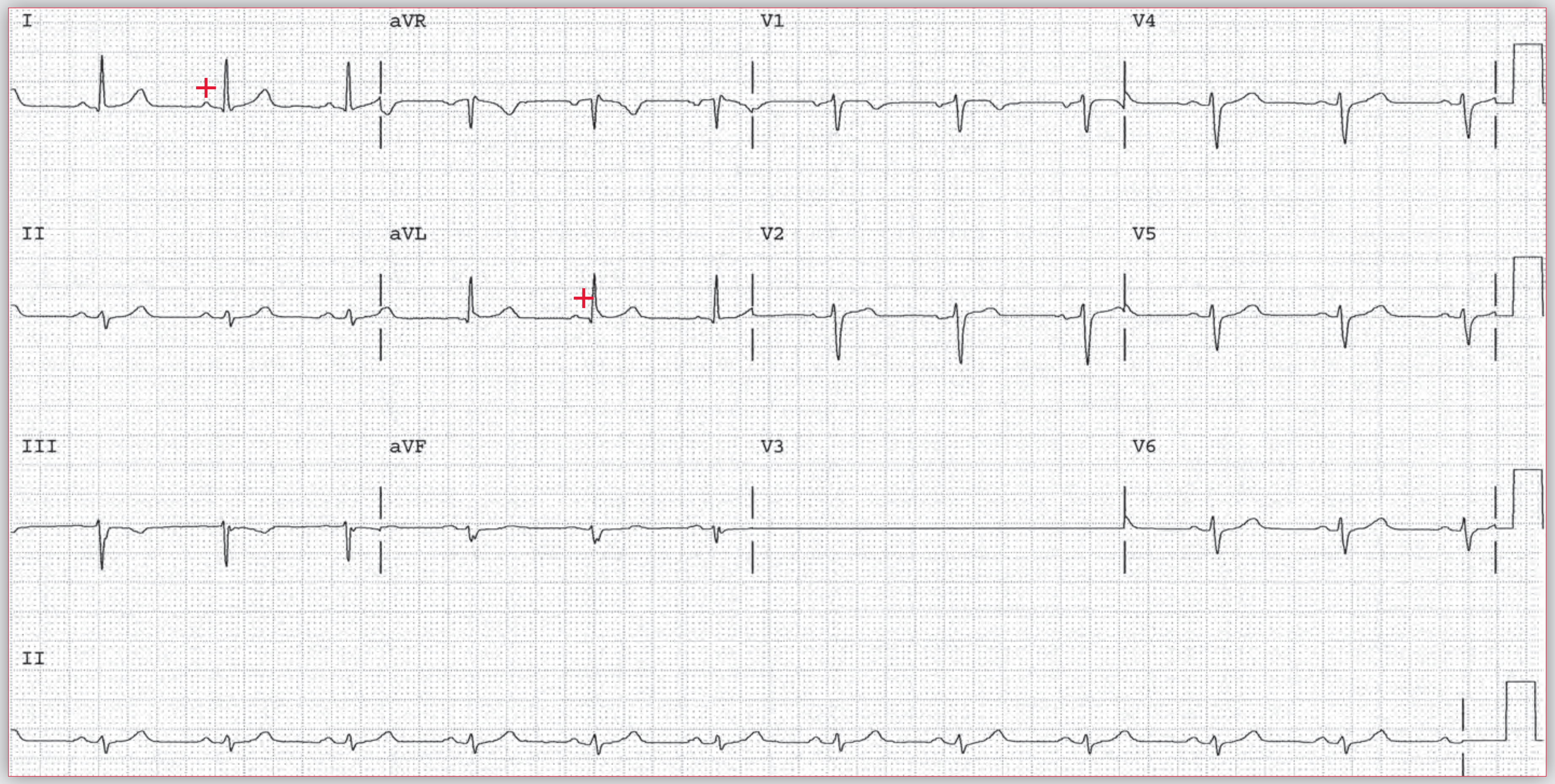


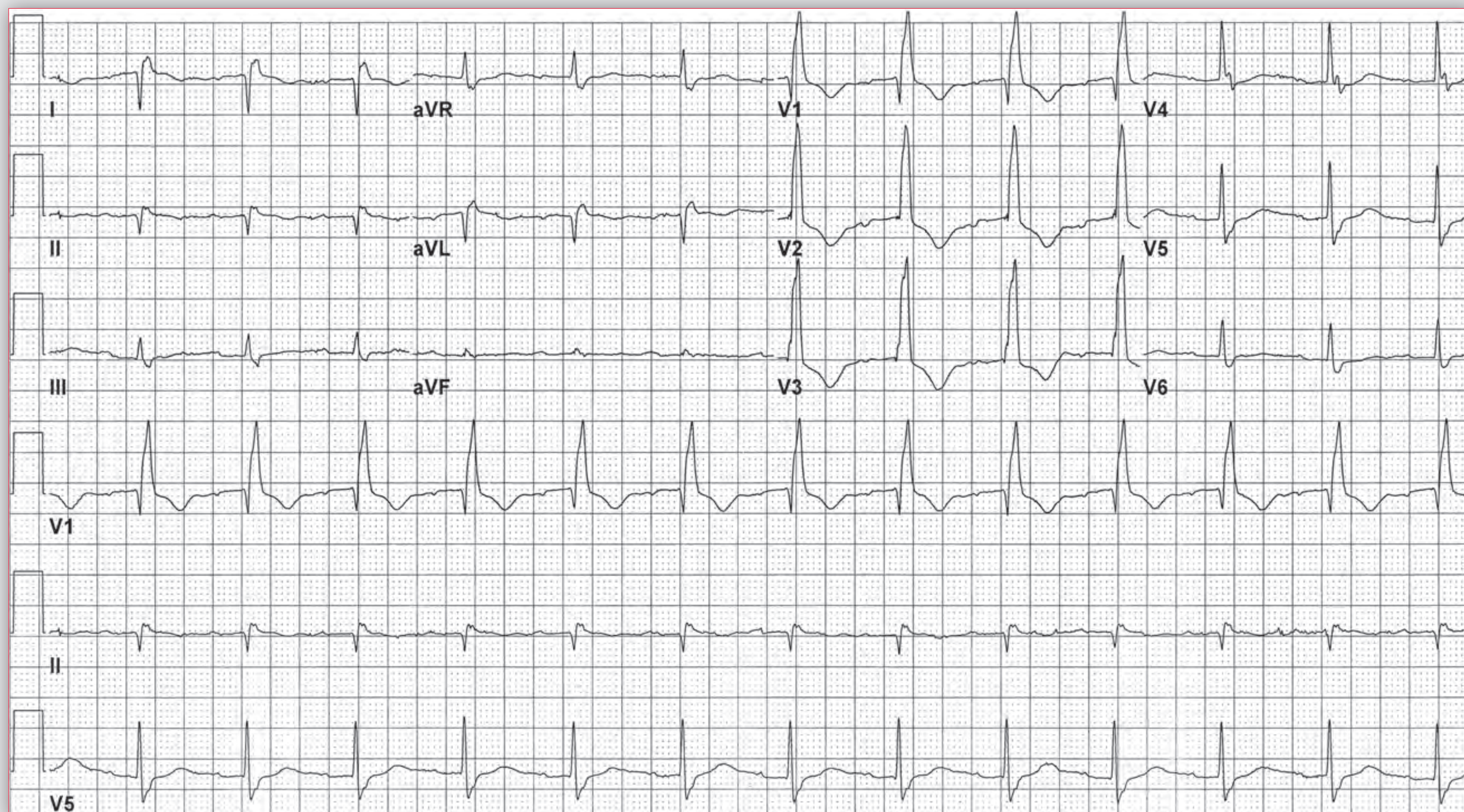
ECG 60B Analysis: Normal sinus rhythm, leads normal

ECG 60B was recorded with the right and left arm electrodes in the correct positive. It can be seen that the QRS complex, P waves and T waves are positive in leads I and aVL (**+**) and are negative in lead aVR. This is a normal pattern and hence there is a sinus rhythm. The QRS complex in lead I while the QRS complex is biphasic in lead II and negative in aVF. Hence the axis is leftward between 0° and –30° (approximately –30°). The QRS complex duration, morphology, and the QT/QTc intervals are the same as seen in ECG 60A. ■

Core Case 61

A 71-year-old woman is seen in her primary care office. An ECG is performed by the medical assistance (ECG 61A) and her physician notices an unusual change in the QRS axis when compared to her baseline ECG (ECG 61B).

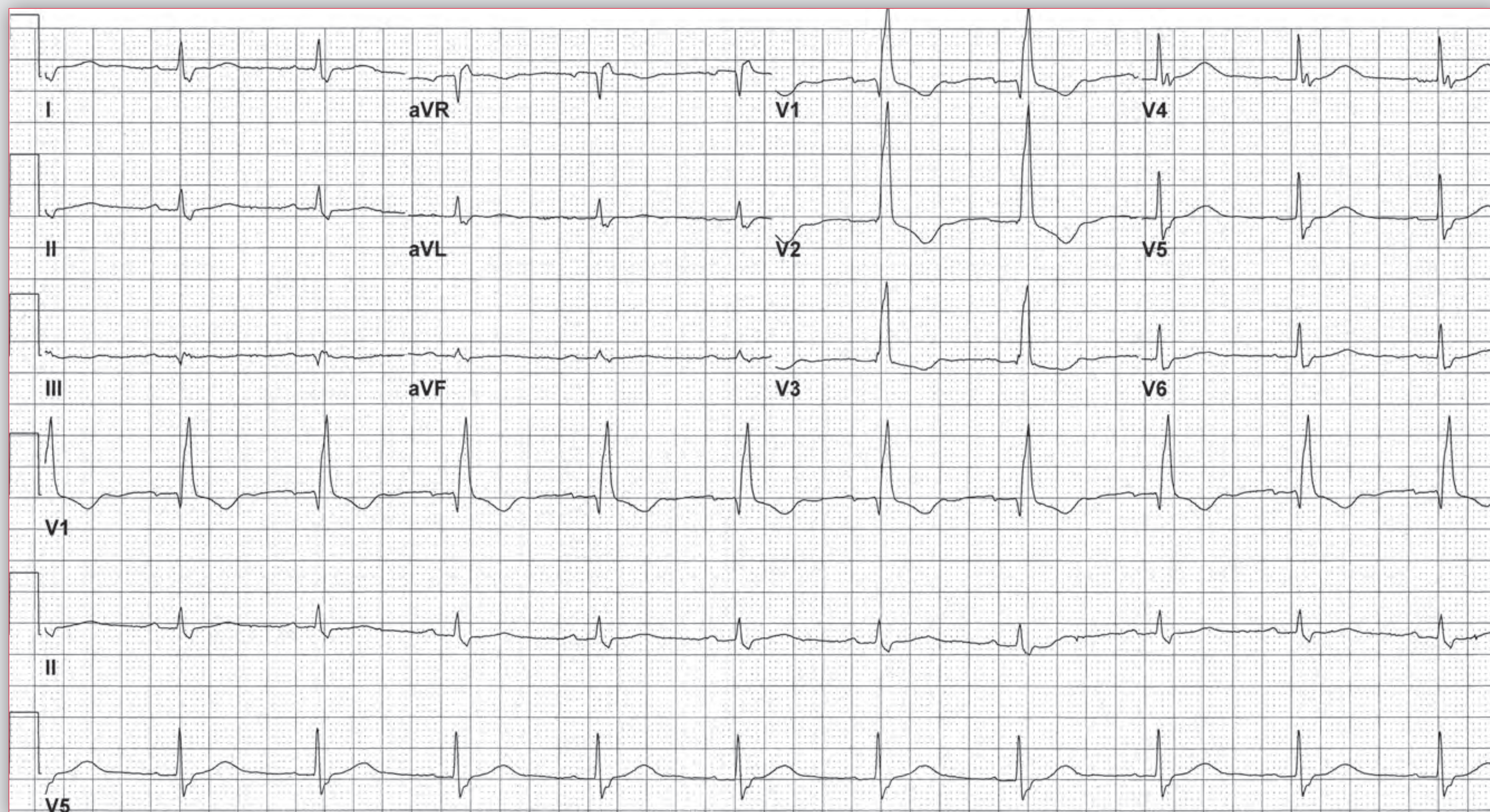
ECG 61A

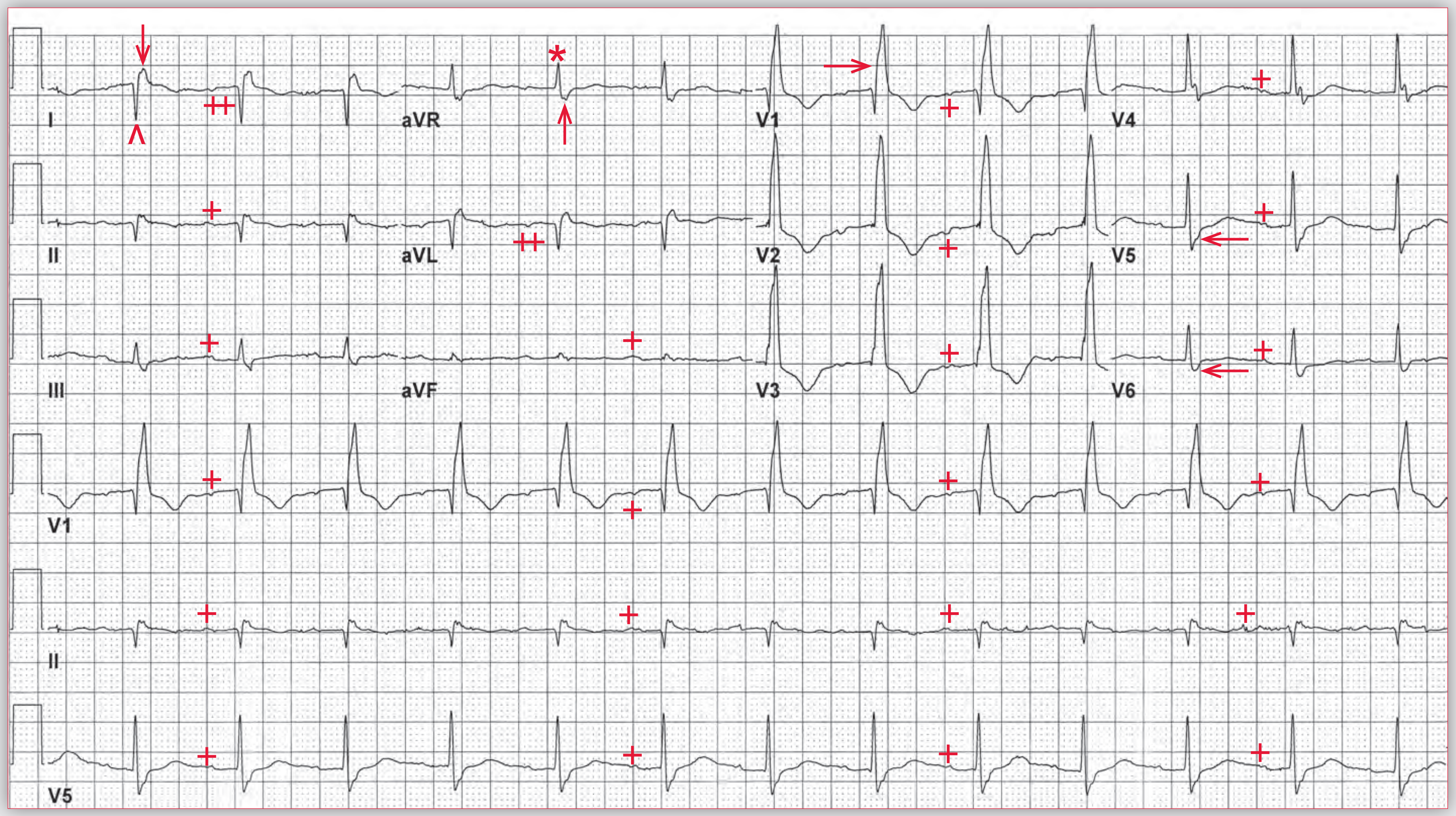


What is a possible explanation for this change?

How can it be corrected?

ECG 61B





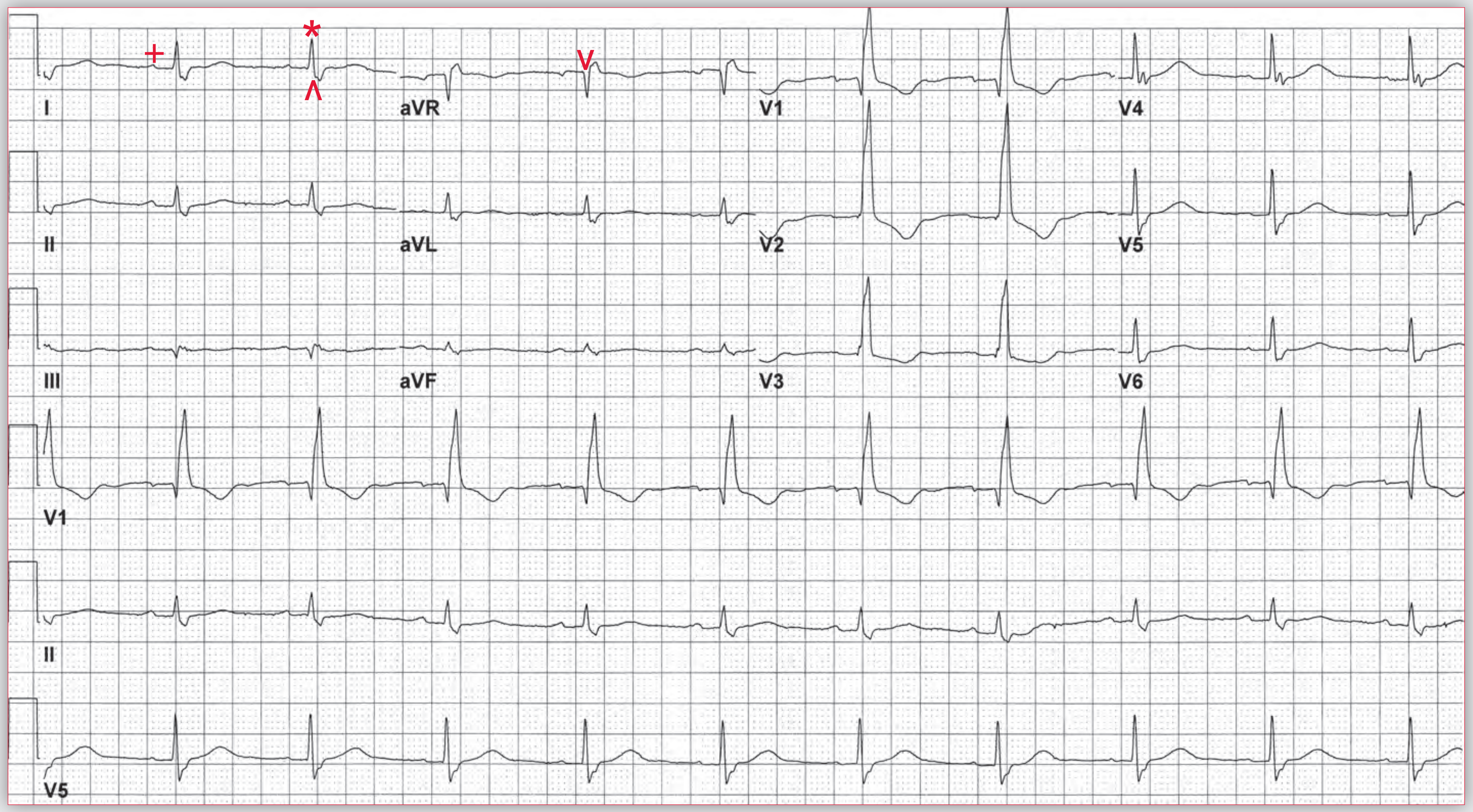
ECG 61A Analysis: Normal sinus rhythm, right bundle branch block (RBBB), indeterminate axis due to right-left arm lead switch

ECG 61A shows there is a regular rhythm with a rate of 80 bpm. There is a P wave (**+**) before each QRS complex with a stable PR interval (0.26 sec). The P wave is negative in leads I and aVL (**++**), although it is positive in leads II, aVF, and V4–V6.

The QRS complex duration is prolonged (0.16 sec), and the morphology resembles a RBBB with a broad terminal R wave in V1 (→) and broad S waves in leads V5–V6 (←). The QT/QTc intervals are prolonged (400/460 msec) but are normal when the prolonged QRS complex duration is considered (340/390 msec). However, in lead I the complex has a Q wave (^) and a broad R wave (↓) while in aVR there is a tall R wave (*****) with a broad S wave (↑). This QRS complex morphology is not typical for a RBBB in these leads. In addition there is a strange and indeterminate QRS axis, with a negative QRS complex in leads I and II and positive in lead aVF. This, therefore, does not fit any particular pattern.

The negative P wave in leads I and aVL (↓), the strange axis and the peculiar QRS morphology in leads I and aVR is characteristic of a right-left arm lead switch, with leads I, aVR, and aVL being upside down.

continues



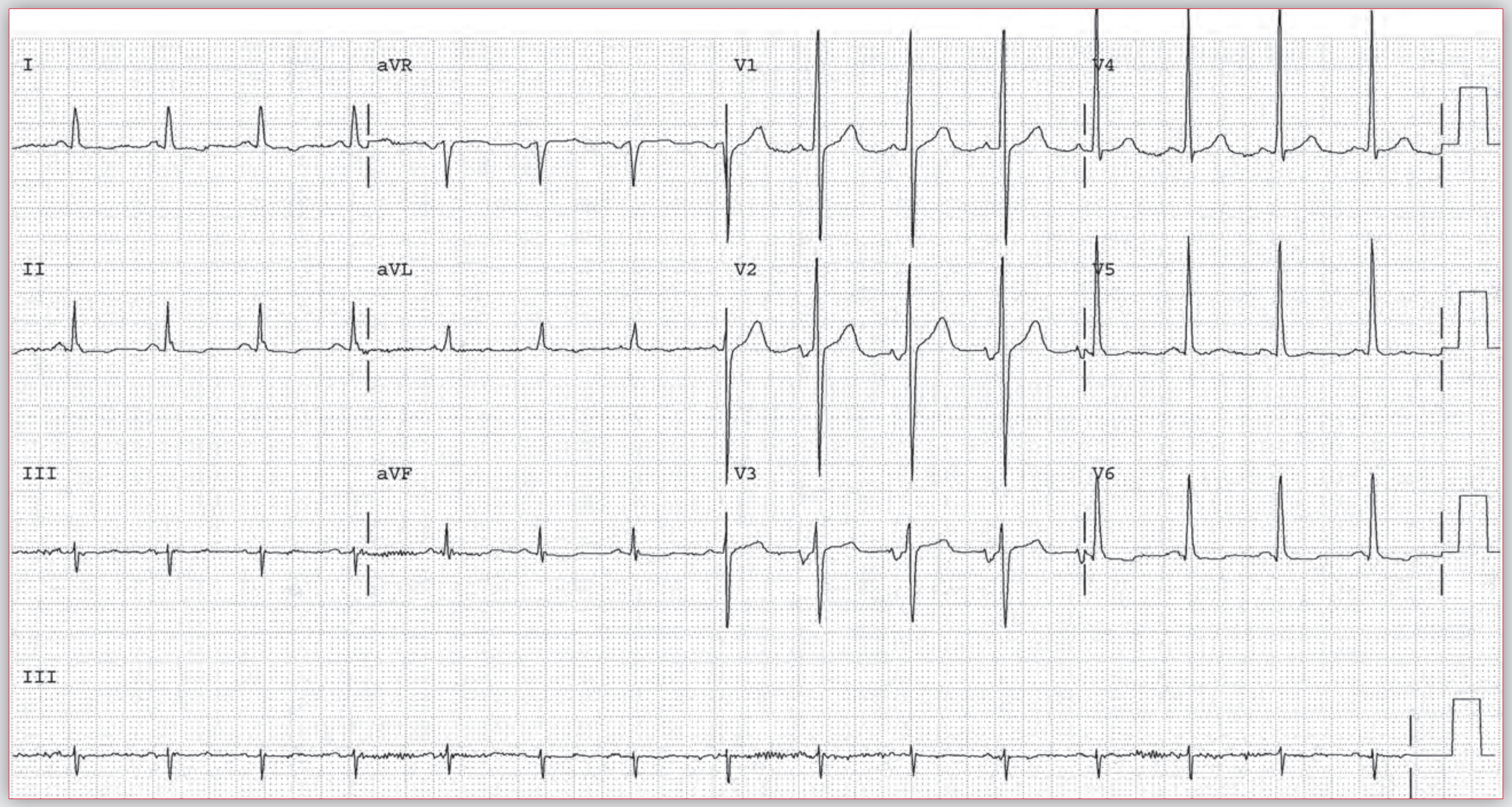
ECG 61B Analysis: Normal sinus rhythm, RBBB, leads placed normally

Indeed, **ECG 61B** was recorded with the right and left arm leads being in the proper position. It can be seen that the QRS morphology is lead I is now typical for a right bundle branch block with an R wave (*) and broad S wave (^), a Qr morphology in aVR (v), a normal axis between 0° and +90° (QRS positive in leads I and aVF) and a positive P wave (+) in leads I and aVL. Therefore, both ECGs show a sinus rhythm.

Core Case 62

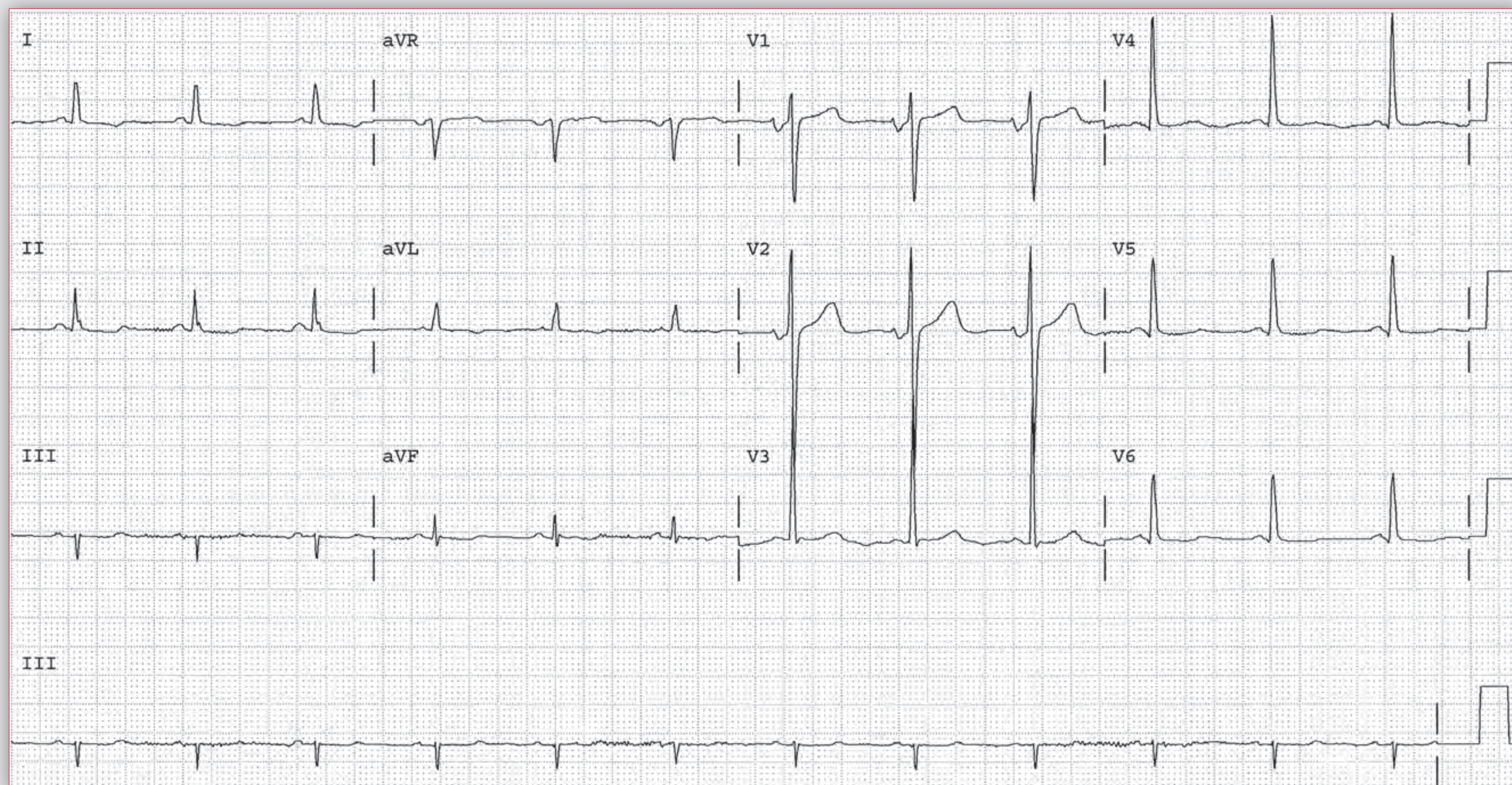
A 29-year-old man is seen at a pre-employment physical exam. He has been healthy and has not complaints. His physician notes a very tall R wave in V1 (ECG 62A). He asks for the ECG to be repeated (ECG 62B).

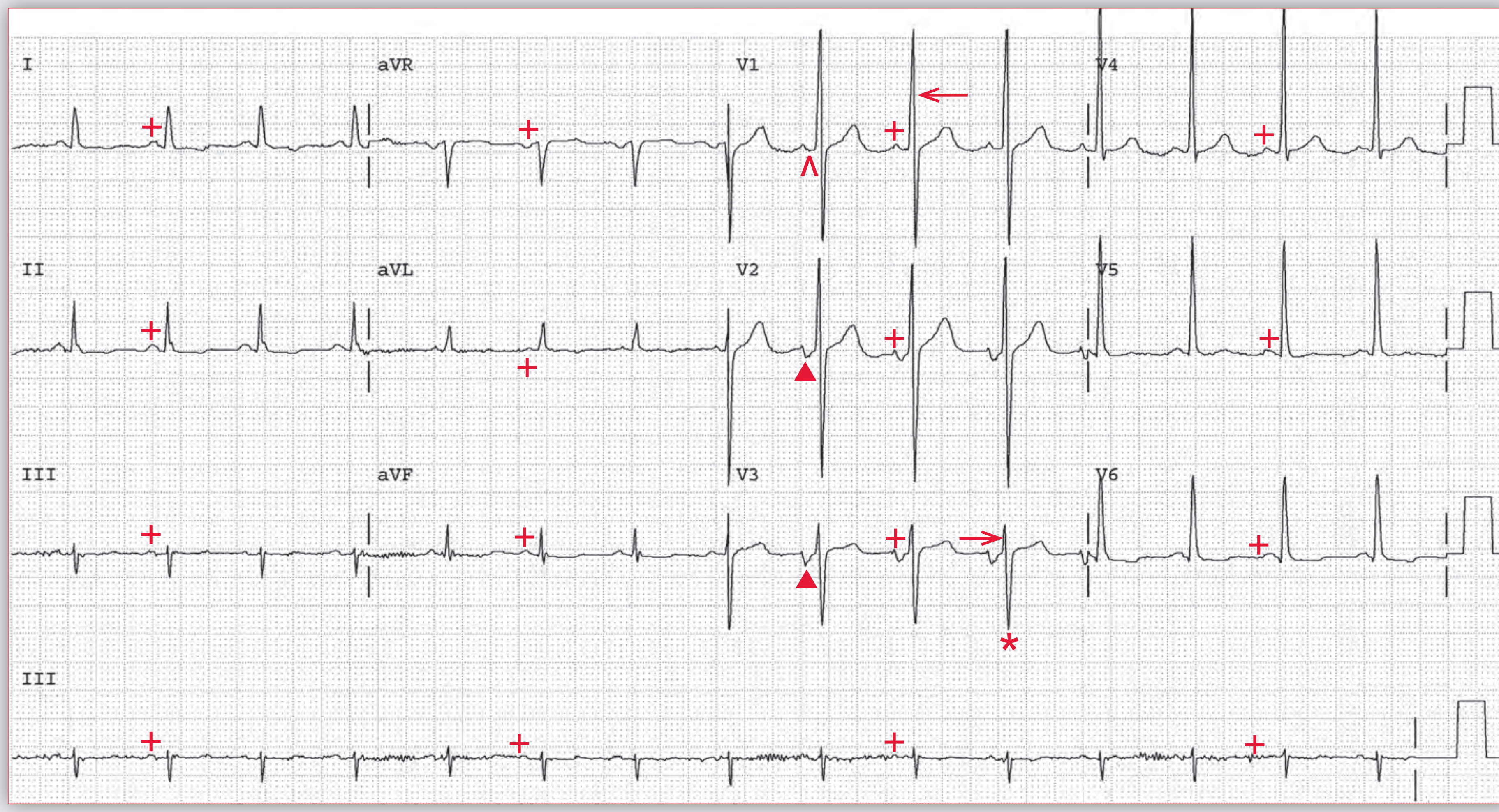
ECG 62A



What is the most likely explanation?

ECG 62B





ECG 62A Analysis: Normal sinus rhythm, V1–V3 lead switch

ECG 62A shows there is a regular rhythm at a rate of 90 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and the axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (360/440 msec).

The R-wave progression across the precordial leads V1–V3 is abnormal. There is a tall R wave in lead V1 (←) that decreases in amplitude to V3, where the R-wave amplitude is decreased (→), but there is a deep S wave (*). Hence this looks like reverse R-wave progression from V1–V3, although the QRS morphology and R waves are normal in leads V4–V6. In addition the P waves in leads V3 and V2 are biphasic (▲), while they are positive (^) in lead V1. Hence the QRS and P-wave morphology in lead V3 is more typical of what is seen in lead V1. Therefore, the QRS and P wave pattern that is seen is the result of a V1–V3 lead switch. Hence the R-wave progression and P wave are normal since V3 is actually V1 and V1 is actually V3.

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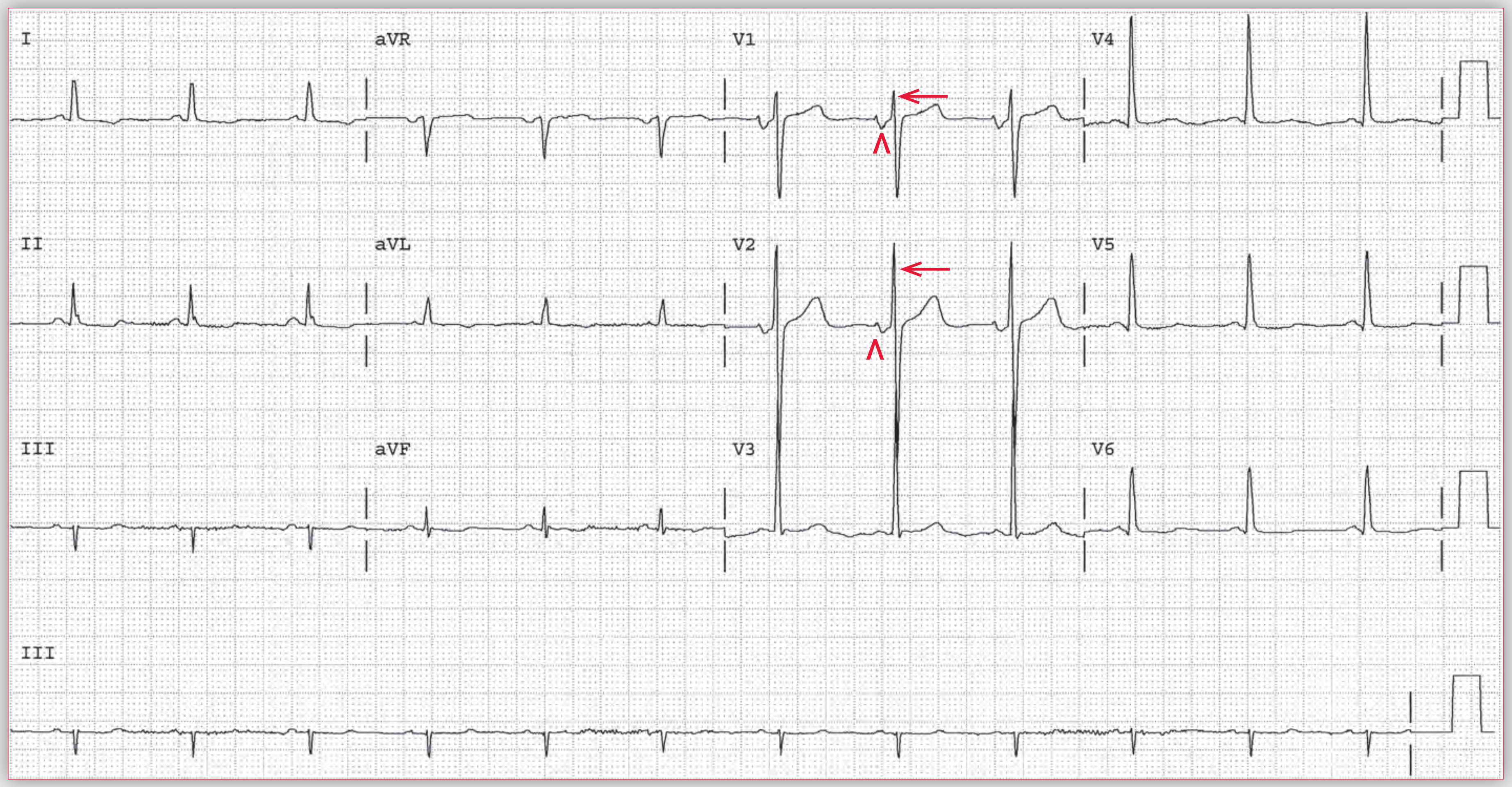


ECG 62B Analysis: Normal sinus rhythm, lead position correct

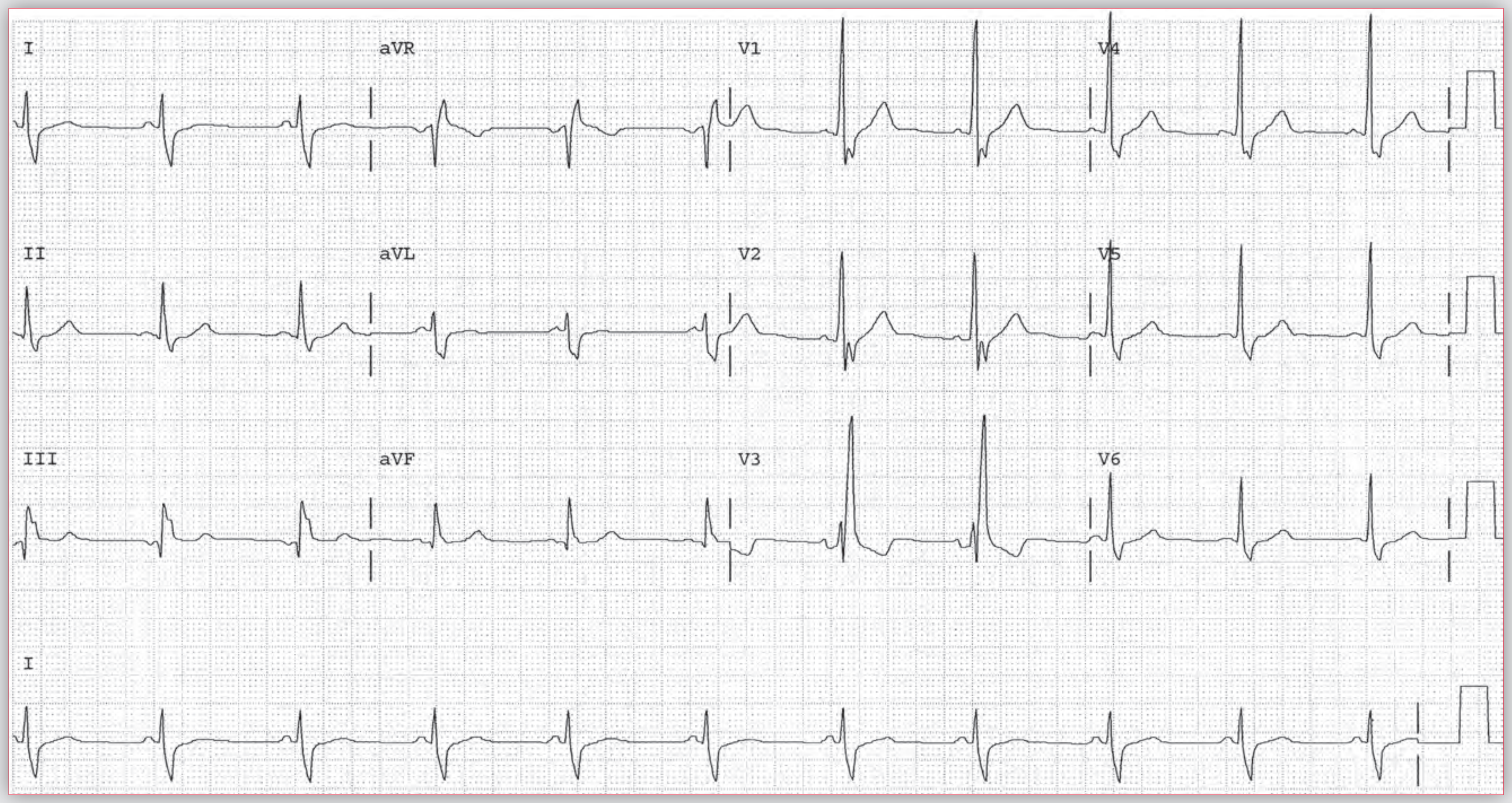
ECG 62B was recorded with the chest leads in the correct position. The P waves in leads V1 and V2 (^) are now biphasic and normal for these leads and the R-wave progression (←) in leads V1–V3 is now normal, *ie*, becomes increasing taller. ■

Core Case 63

A 56-year-old man with known pulmonary sarcoidosis presents for routine evaluation. He states he is well with stable dyspnea that limits his ability to climb steps or hilly inclines. During his exam, an ECG is obtained (ECG 63A). The primary doctor interprets the ECG and is concerned about severe right ventricular hypertrophy secondary to pulmonary hypertension.

The physician consults a cardiologist colleague and requests a repeat ECG (ECG 63B).

ECG 63A



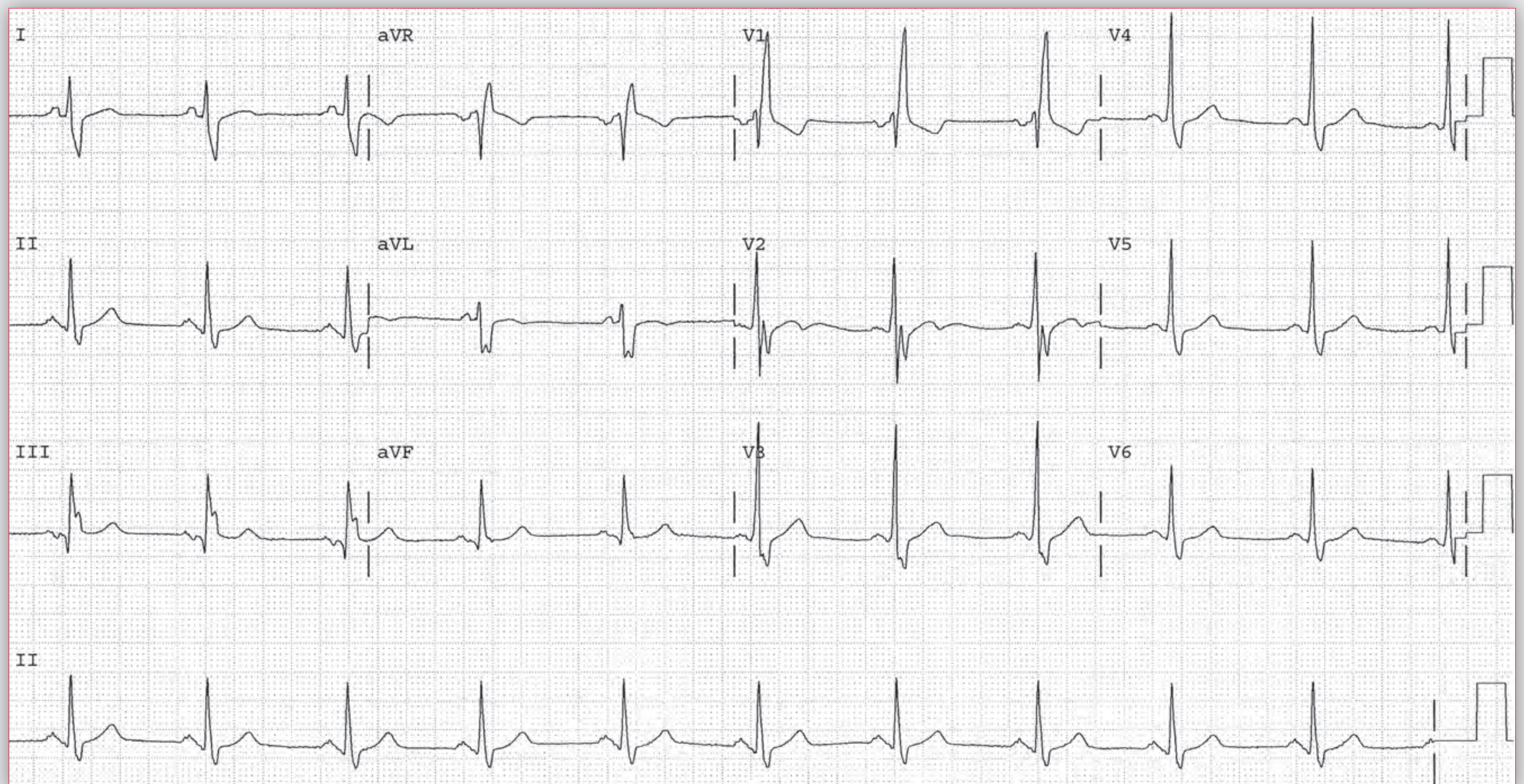
What findings on ECG 63A prompted this concern?

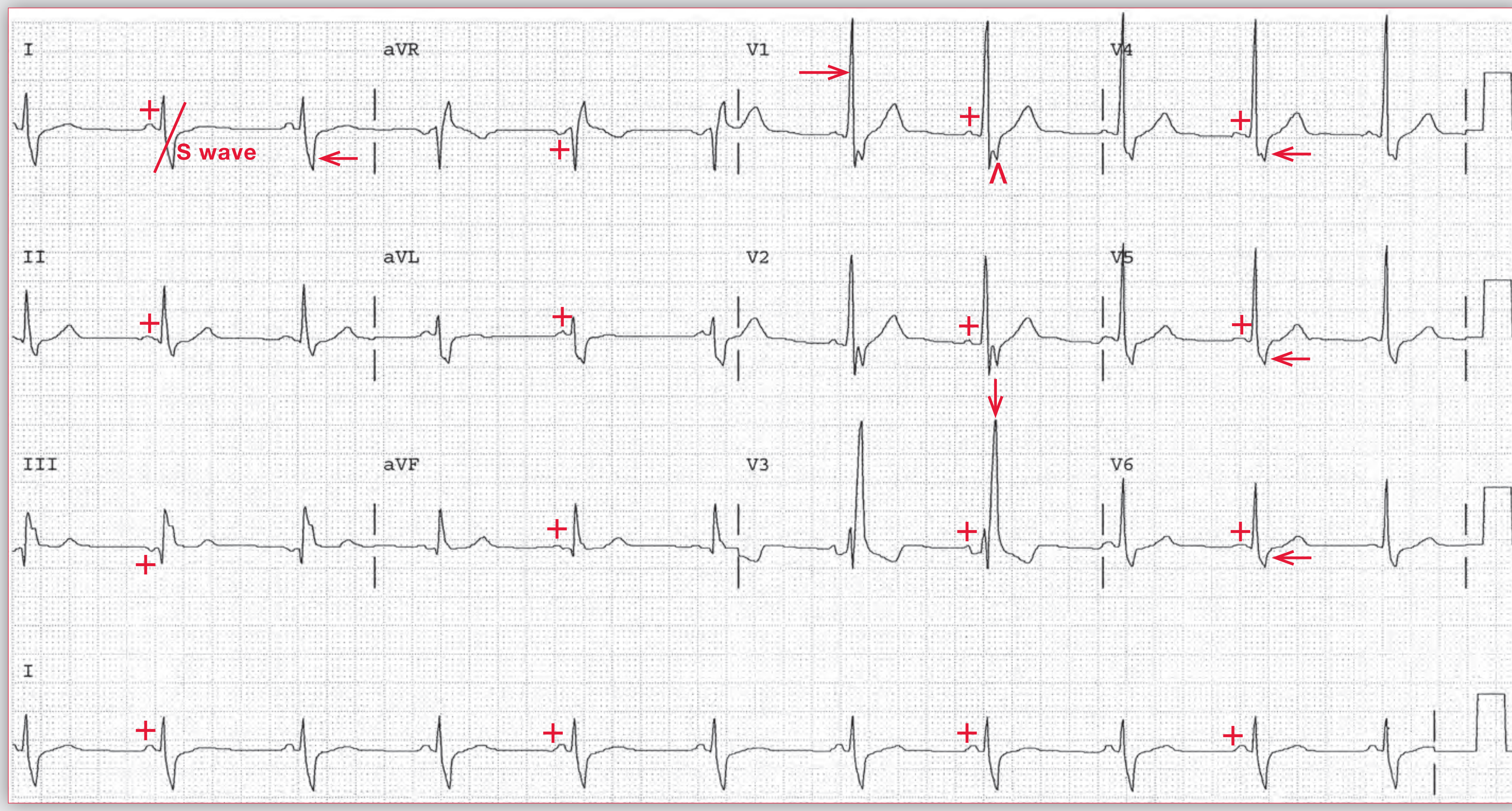
Comparing the two tracings, what has changed?

What is the reason for this change?

Based on analysis of ECG 63B, how would you re-interpret ECG 63A?

ECG 63B





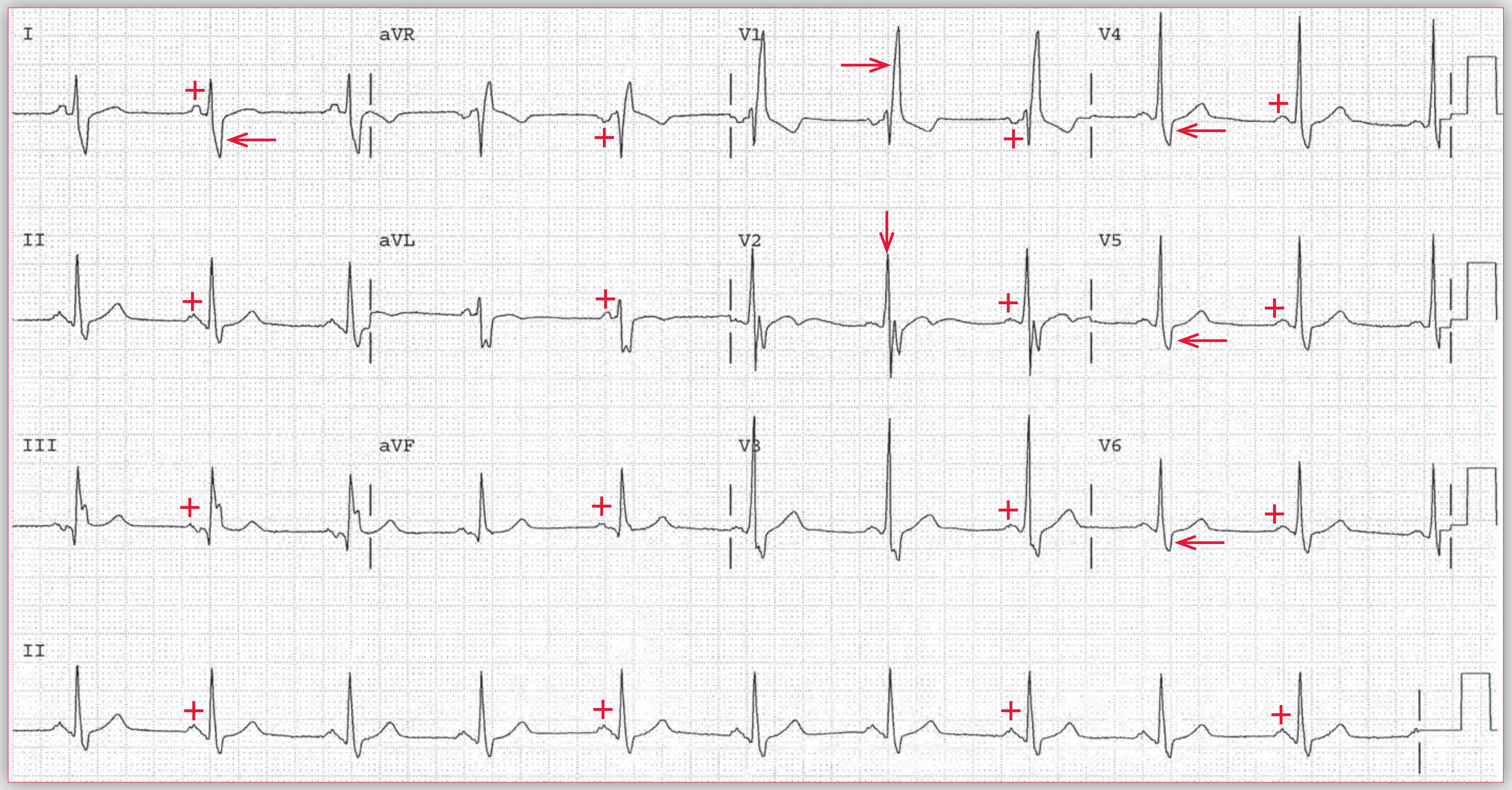
ECG 63A Analysis: Normal sinus rhythm, right bundle branch block, V1–V3 lead reversal

There is a regular rhythm with a rate of 64 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6, and this is a sinus rhythm.

The QRS complex duration is prolonged (0.16 sec). There is a broad S wave in leads I and V4–V6 (←), suggesting a right bundle branch block (RBBB) However, the QRS morphology in lead V1 is not typical of a RBBB as there is a tall R wave (→) and broad S wave (^) rather than an RSR′ complex. In contrast, the QRS morphology in lead V3 is typical for a RBBB as there is an RSR′ morphology (↓) in this lead. Thus this is the result of a V1–V3 lead switch. Noted is reverse R-wave progression in leads V1 to V3 as lead V1 is actually V3, and V3 is actually V1. In addition, the P wave in lead V3 is biphasic, typical for the P wave in lead V1 and it is positive in lead V1, which is typical for the P wave in lead V3. The QT/QTc intervals are prolonged (440/450 msec) but are normal when the prolonged QRS complex duration is considered (380/390 msec).

The axis in the frontal plane is normal between 0° and +90° (QRS positive in leads I and aVF). The QRS complex in lead I appears to be more negative than positive as the result of the broad S wave which represents delayed right ventricular activation from the right bundle branch block. The S wave is ignored when determining the axis that is based on the direction of left ventricular activation.

continues



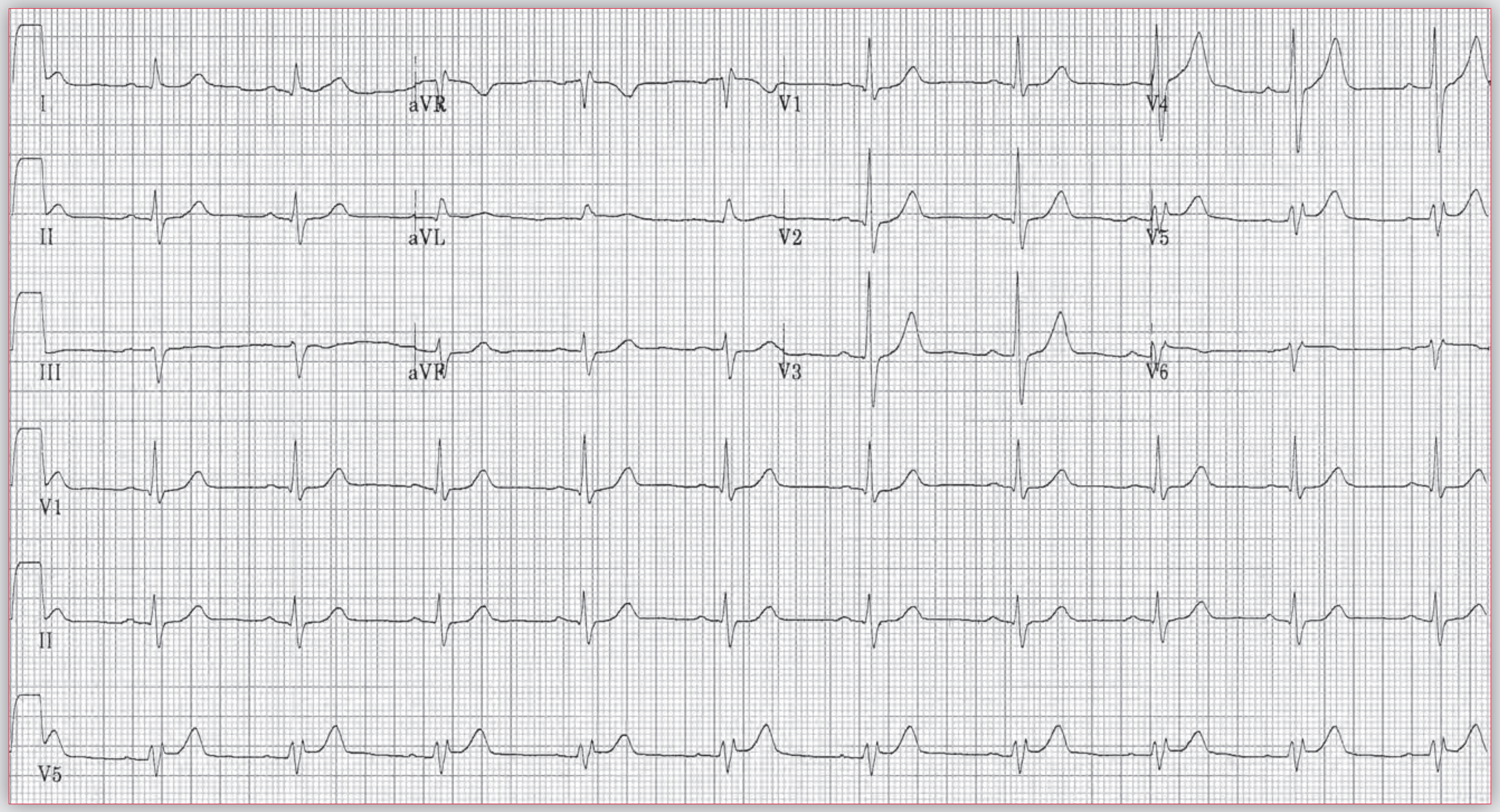
ECG 63B Analysis: Normal sinus rhythm, RBBB, lead position normal

ECG 63B represents the recording obtained with the precordial leads in the correct location. There is a P wave (+) before each QRS complex, and it has the same morphology and the PR interval is the same as seen in ECG 63A. The QRS complex duration and axis and the QT/QTc intervals are the same as in ECG 63A. The QRS complex morphology is now normal in lead V1–V3 and it can be seen that there indeed is a typical RBBB morphology of the QRS complexes in leads V1–V6; *ie*, RSR′ in V1 (→) and broad S wave in leads I, V4–V6 (←). There is early transition with a tall R wave in lead V2 (↓). This is due to counterclockwise rotation in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are seen earlier in there precordial leads, and hence there is a tall R wave in lead V2. This is not diagnostic of right ventricular hypertrophy or a posterior wall myocardial infarction, which has a tall R wave in lead V1. Indeed, abnormalities of the right ventricle, particularly right ventricular hypertrophy, cannot be diagnosed in the presence of a RBBB as right ventricular activation is not via the normal His-Purkinje system, but is directly via the ventricular myocardium and hence not normal. ■

Core Case 64

A 48-year-old man with a history of hypertension and a previous myocardial infarction is seen by his primary care provider for a routine physical examination. He has no other medical history, and his only medication is an ACE inhibitor. An ECG is obtained (ECG 64A). His physician compared this ECG to the one obtained 1 year before (ECG 64B). He becomes concerned about a marked change.

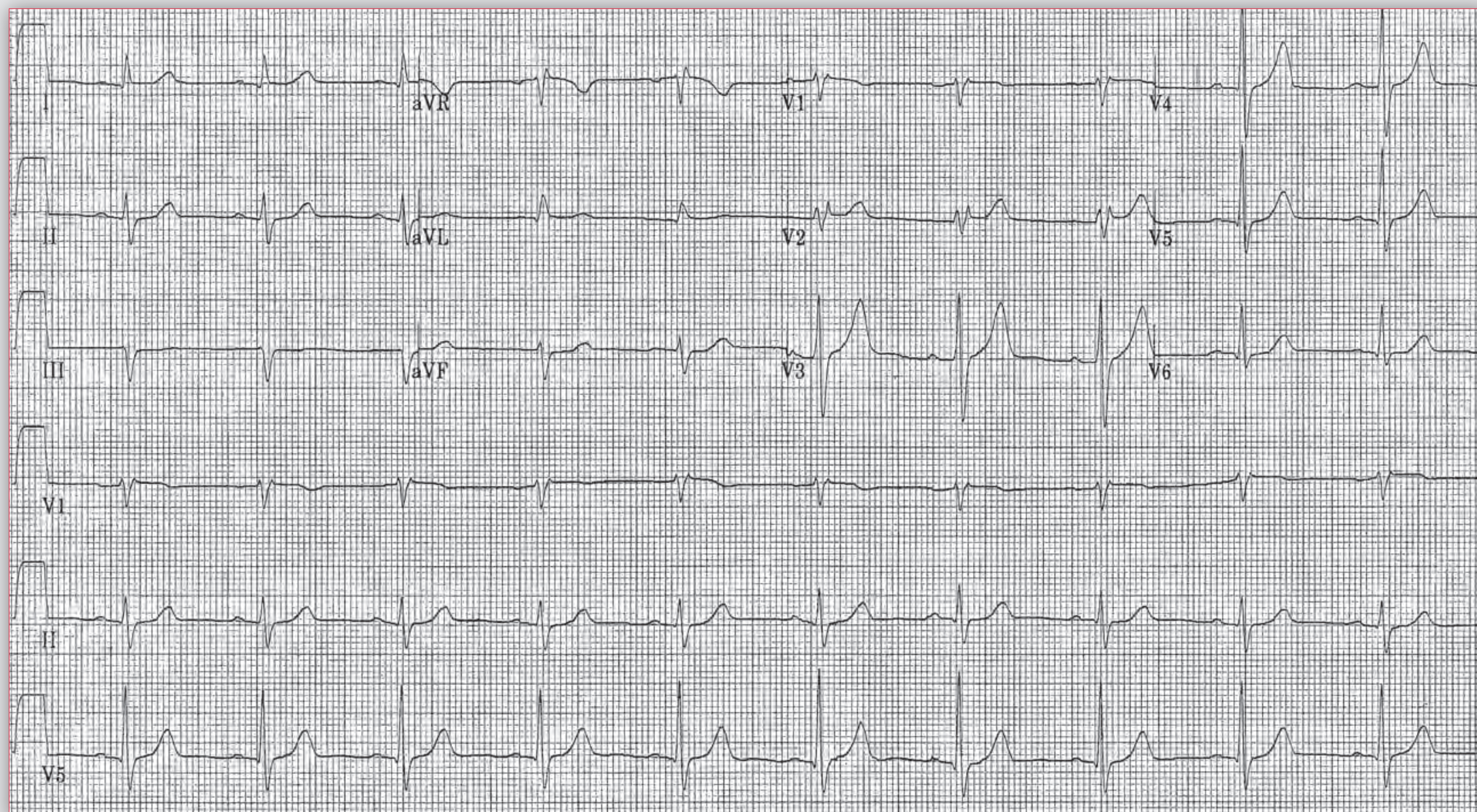
ECG 64A

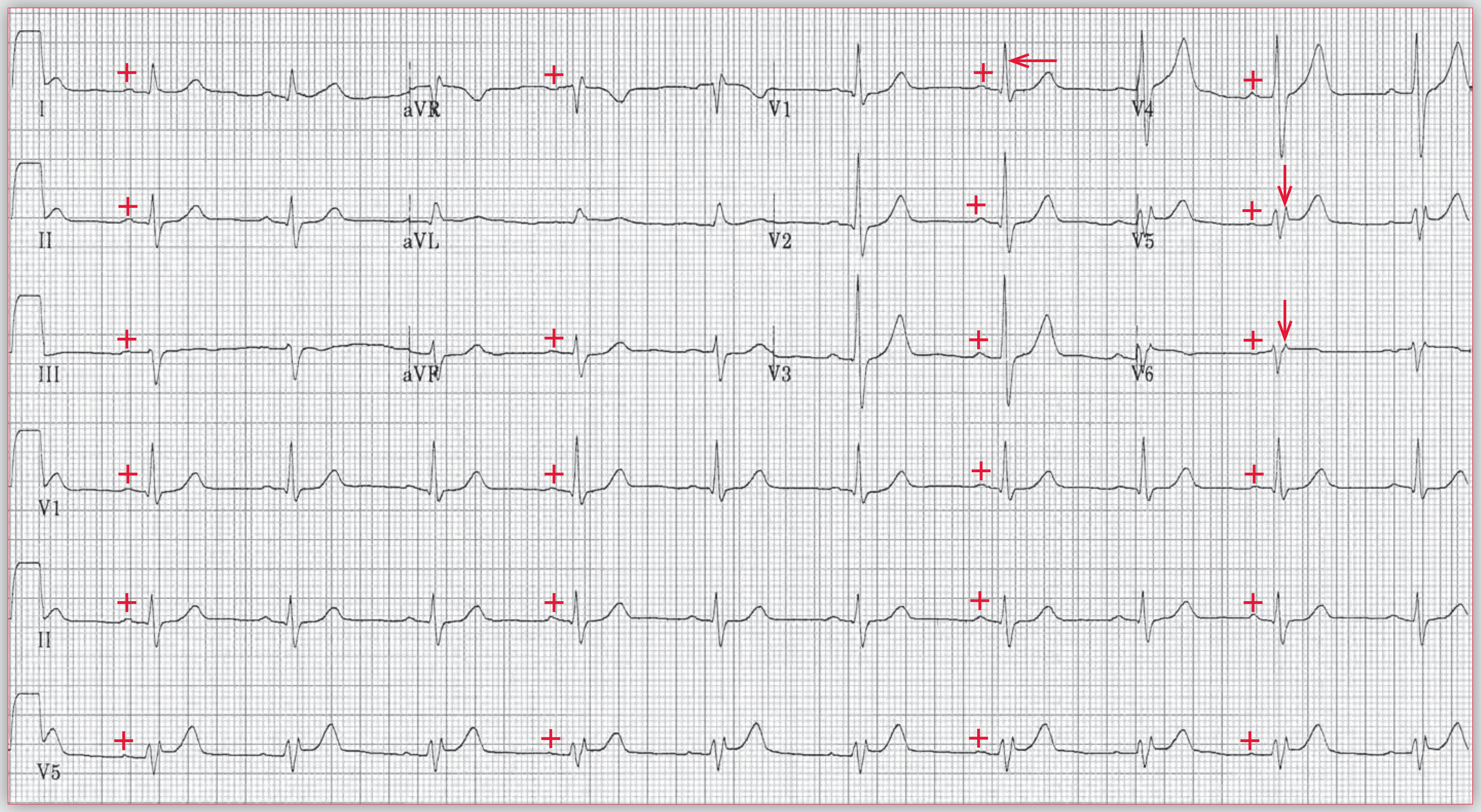


What abnormality is seen on the ECG?

What accounts for the change from baseline?

ECG 64B





ECG 64A Analysis: Normal sinus rhythm, tall R wave in V1 with reverse R-wave progression (V1 through V6 lead misposition), right-sided leads

ECG 64A shows there is a regular rhythm with a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Therefore, this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec), and there is a normal axis of about 0° (positive in leads I and isoelectric in lead aVF). The QT/QTc intervals are normal (420/420 msec).

There is a tall R wave in lead V1 (←), which is abnormal. In addition, there is an RSR′ complex in leads V5–V6 (↓), which is also unusual as this pattern is more typically seen in leads V1–V2. There are a number of etiologies for a tall R wave in V1, including:

1. Right ventricular hypertrophy
2. Posterior wall myocardial infarction
3. Wolff-Parkinson-White pattern
4. Hypertrophic cardiomyopathy
5. Early transition
6. Dextrocardia
7. Duchenne's muscular dystrophy
8. Lead malposition
9. Right-sided leads

It should be noted that there is also reverse R-wave progression, *ie*, the R-wave amplitude becomes progressively shorter from V1–V6. This is suggestive of either dextrocardia or lead misplacement of V1–V6 or right-sided leads. As the axis is normal and the P waves are positive in lead I, this is not dextrocardia. Hence the etiology for this abnormality is V1–V6 lead misplacement or right-sided leads.

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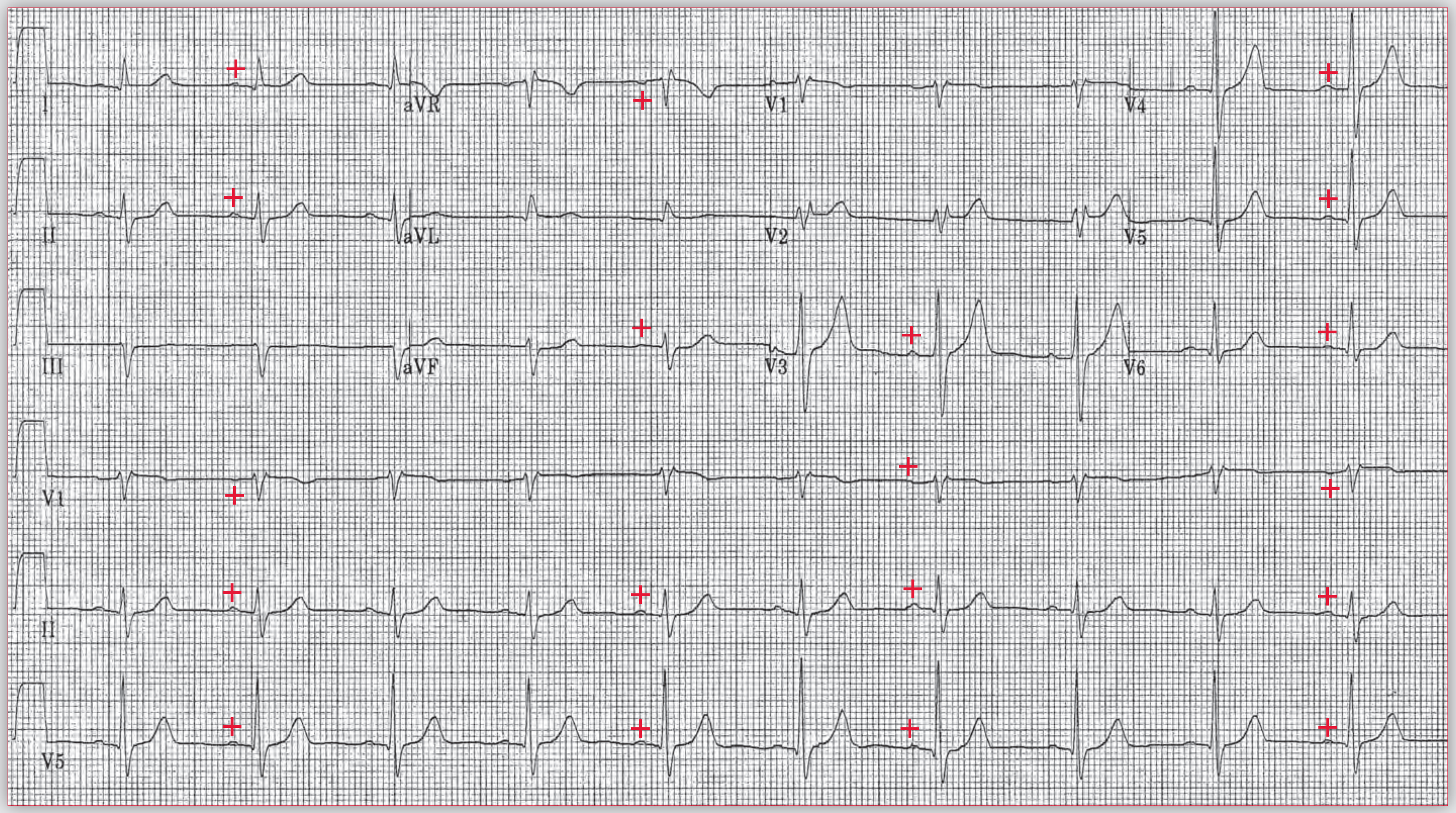


ECG 64B Analysis: Normal sinus rhythm

ECG 64B is the patient's baseline ECG and is identical to an ECG repeated after ECG 64A was obtained. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). This is a normal sinus rhythm. The QRS complex duration and axis are the same as ECG 64A. However, there is now a normal QRS complex in V1 with a normal small R wave. The R-wave progression from V1–V6 is now normal. In addition, there is an RSR′ complex in V1–V2, which is identical in morphology to what was seen in V5–V6 in ECG 64A, confirming the fact that ECG 64A was recorded with V1–V6 leads in the wrong locations. Hence this ECG was recorded with the chest leads in the right position. Since the QRS complex duration is not prolonged, the R′ in V1–V2 represents a right ventricular conduction delay. ■

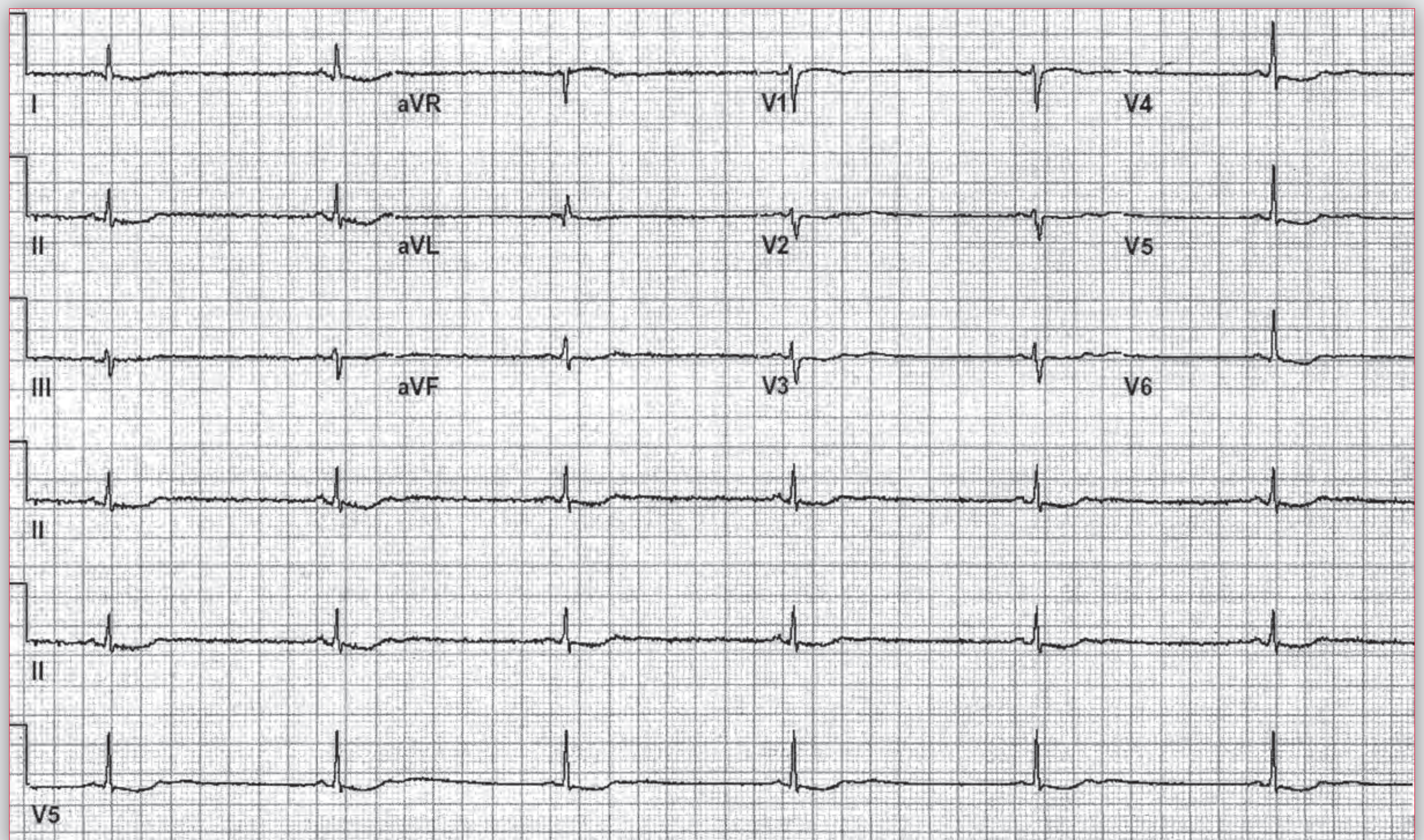
Notes

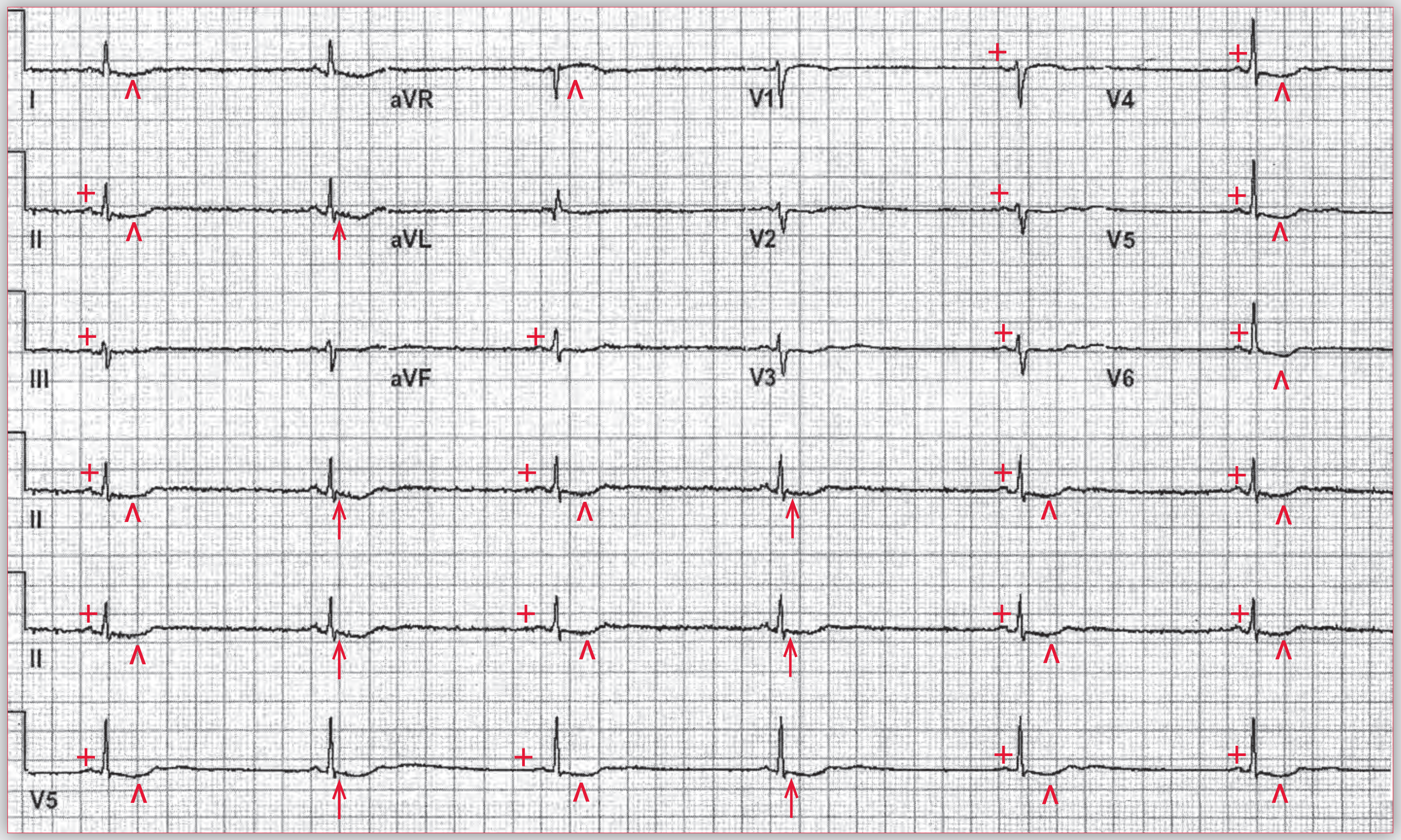
Core Case 65

An 81-year-old man is seen in the emergency department for complaints of sharp, pleuritic chest pain. Although the pain is felt to be noncardiac, an ECG is obtained, and the emergency department physician is concerned about what it shows. Although the patient does not have a history of coronary artery disease, he does have a history of paroxysmal atrial fibrillation for which he has been taking digoxin for many years.

What is the abnormality of concern?

What is the likely etiology?





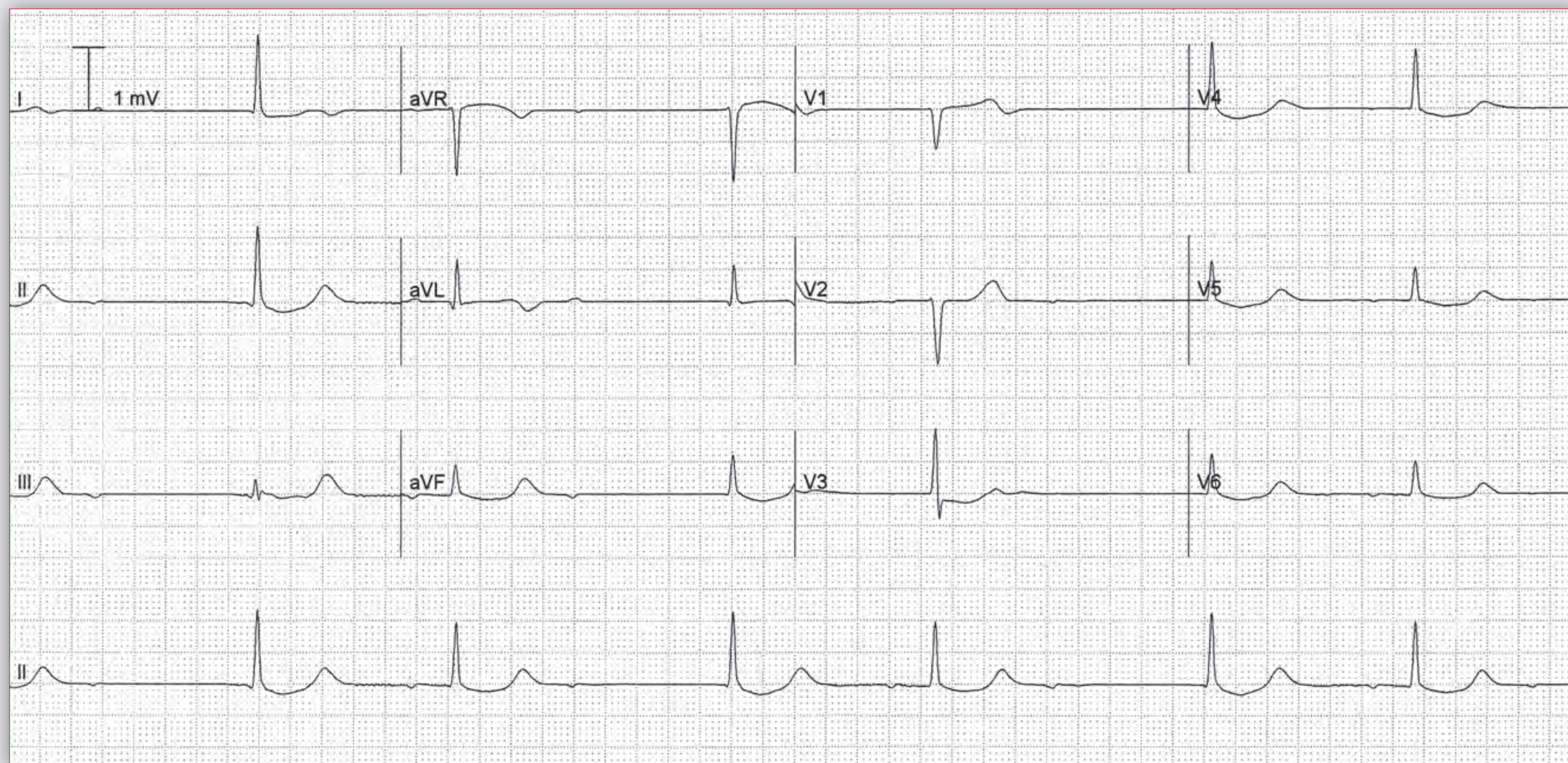
ECG 65 Analysis: Sinus bradycardia with sinus arrhythmia, short PR interval, ST segment abnormalities (digoxin effect)

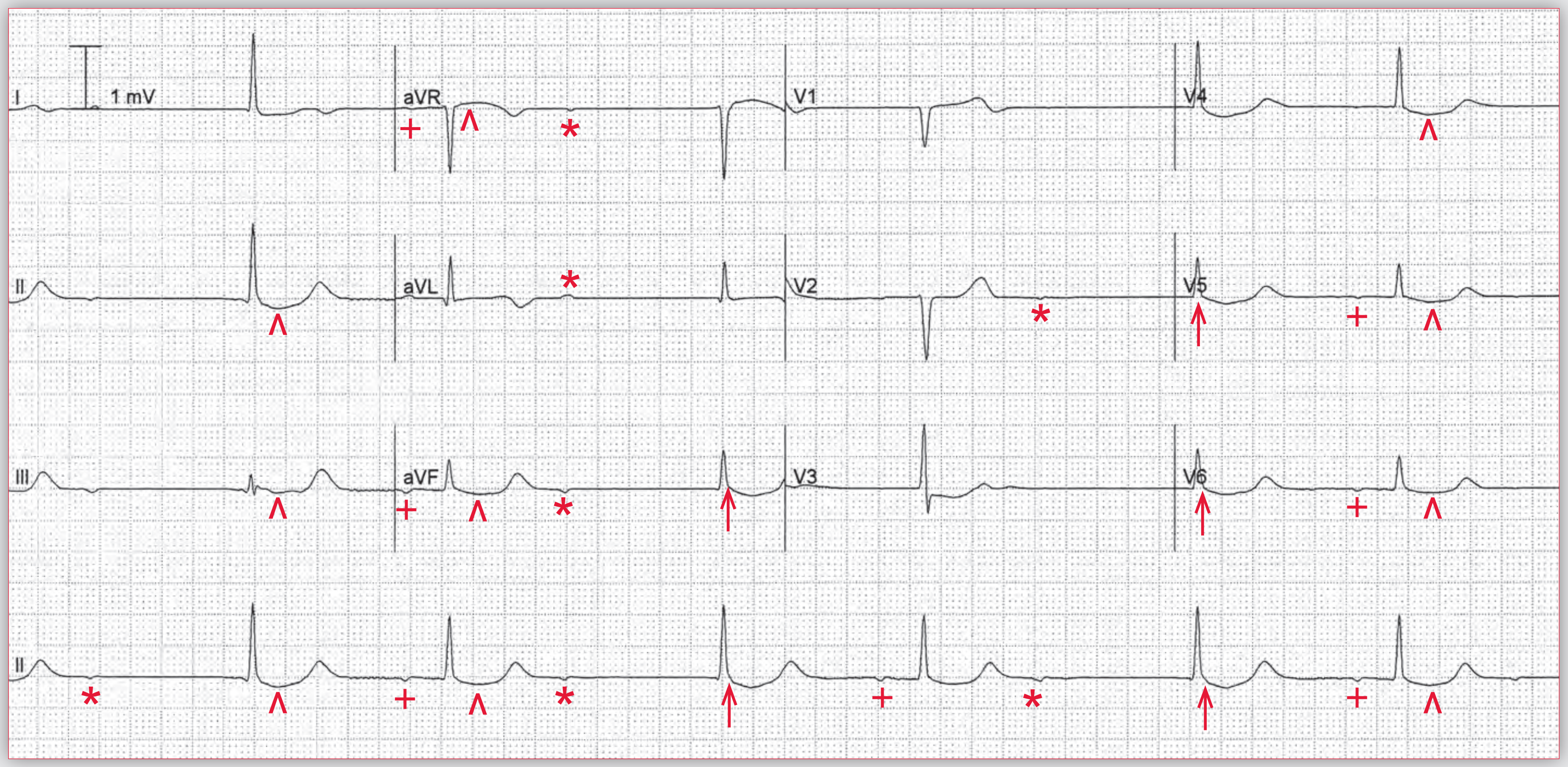
There is a slightly irregular rhythm at a rate of 36 bpm. There is a P wave (+) before each QRS complex with a stable P-wave morphology and PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus arrhythmia. The QRS complex interval is normal (0.08 sec), and there is a normal axis between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (480/370 msec). Noted is sagging, hammock-like, or scooped ST-segment depression (^) with a J point that is at baseline (↑). The ST-segment changes are typical of digoxin and represent a digoxin effect. Digoxin toxicity is defined by the occurrence of arrhythmias as well as clinical symptoms associated with this drug, *ie*, weakness, nausea, vomiting, abdominal discomfort, and visual disturbances (primarily yellow–green halos). ■

Notes

A 74-year-old man on digoxin for rate control of paroxysmal atrial fibrillation is admitted with pre-syncope.

What is the etiology of his lightheadedness?





ECG 66 Analysis: Atrial rhythm with 2:1 AV block with a junctional escape, digoxin effect, possible digoxin toxicity

The rhythm is regularly irregular with an average rate of 36 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (500/390 msec). Noted is sagging ST-segment depression (^) with a J point that is at baseline (↑). These ST-segment changes (sagging, hammock-like, or scooping) are typical of digoxin and represent a digoxin effect.

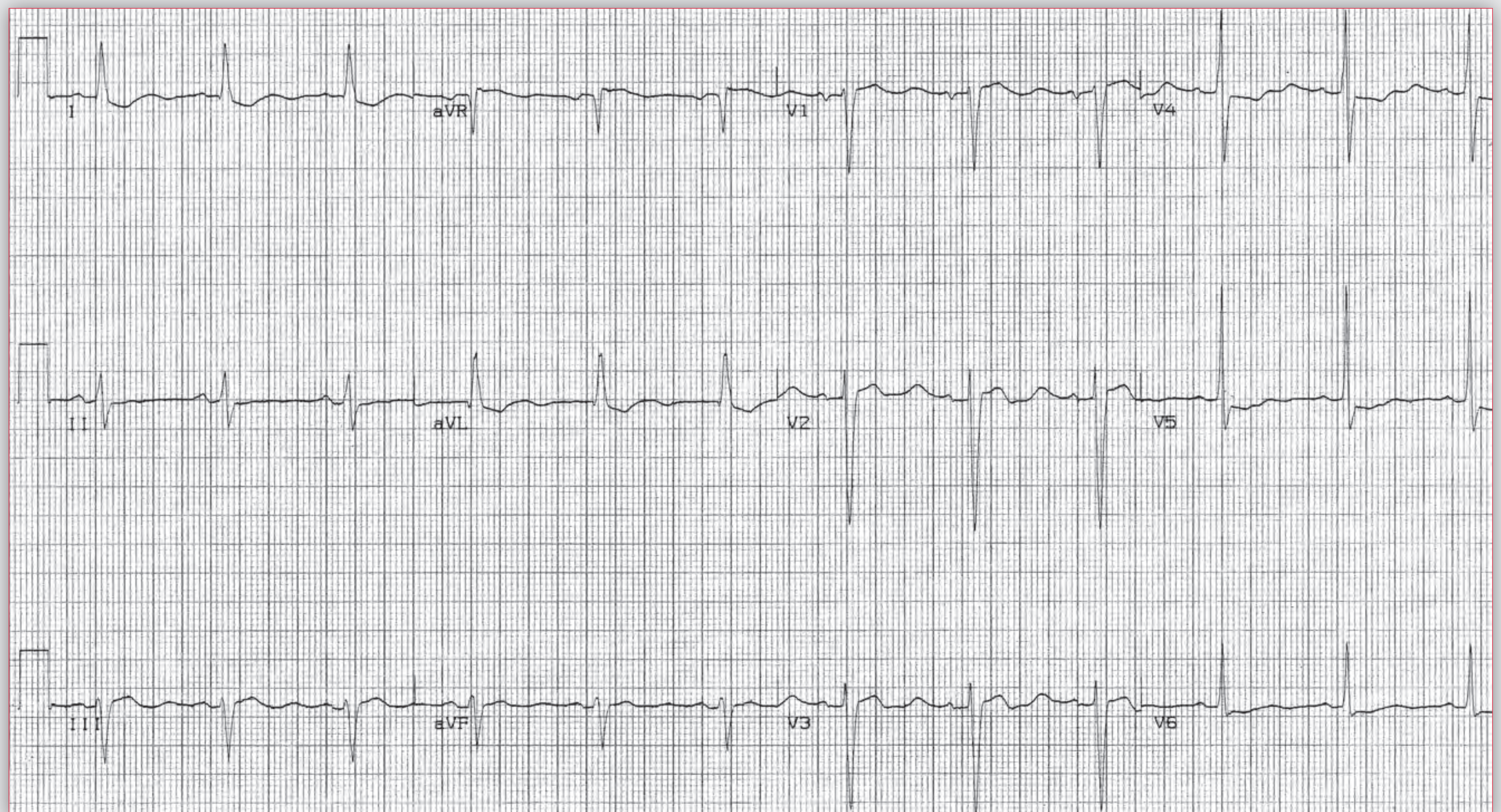
There is evidence of regular atrial activity, at a rate of 58 bpm. The P waves (+) are negative in leads II, aVF, and V5–V6. Hence this is an atrial rhythm. The second, fourth, and sixth complexes are associated with a stable PR interval (0.26 sec). Following the second and fourth QRS complexes, there is an on-time but nonconducted P wave (*), *ie*, there is no QRS complex associated with them. Therefore, this is a second-degree AV block. Following the nonconducted P wave, there is a QRS complex (↑) that does not have a P wave before it and occurs after a pause of 1.8 sec. This QRS complex has the same morphology as the conducted QRS complexes. This is a junctional escape complex that occurs before the next on-time P wave would occur. Hence this is 2:1 AV block with a junctional escape. The 2:1 AV block is a Mobitz type I, related to a conduction abnormality in the AV node. This AV block is likely the result of the increased vagal effect from digoxin. Along with the ST segment abnormalities, this ECG represents digoxin excess and probably digoxin toxicity (*ie*, an atrial tachycardia with second-degree AV block, *ie*, Mobitz type I). ■

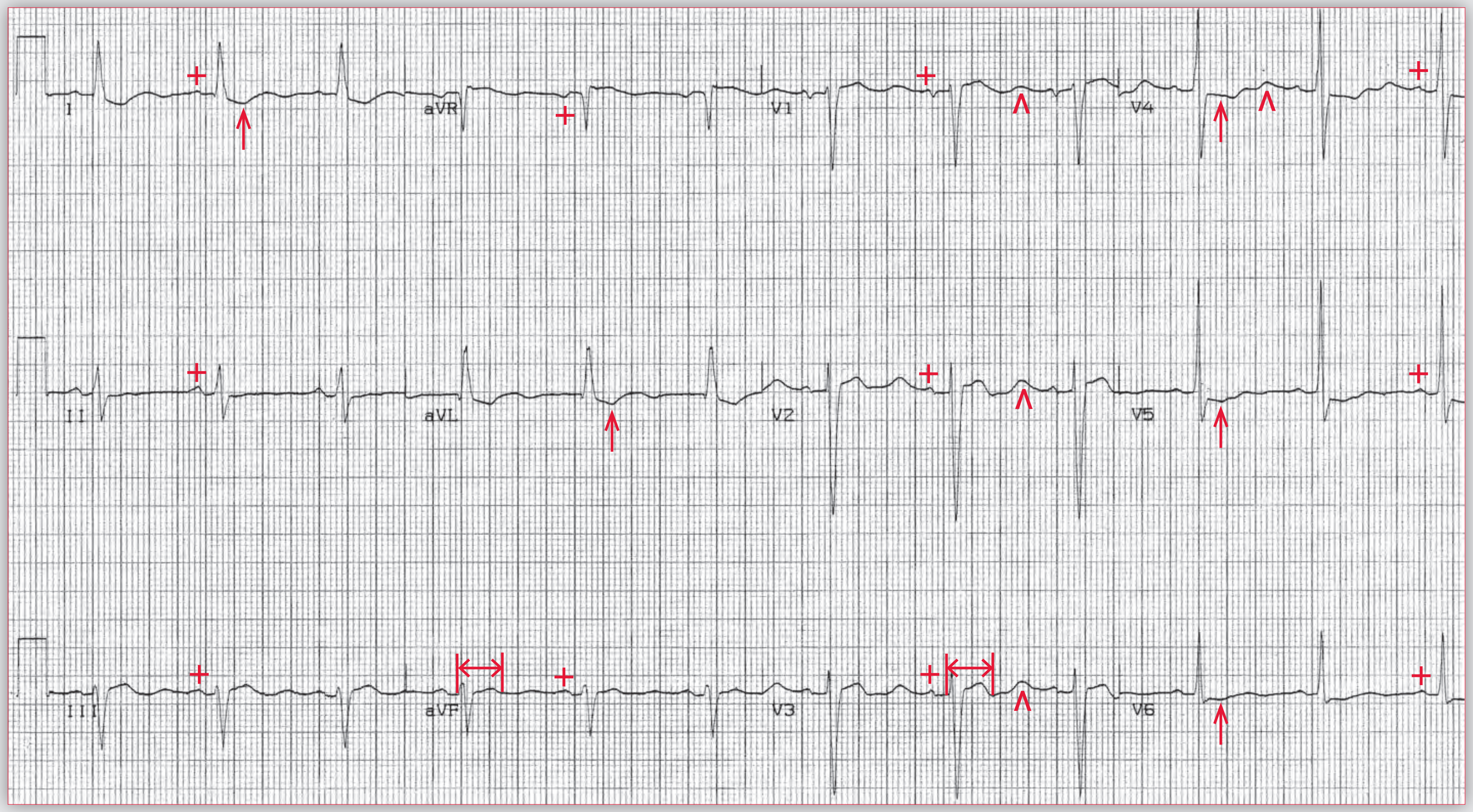
Notes

Core Case 67

A 72-year-old man with congestive heart failure is seen in the office for routine follow up. His ECG demonstrates several abnormalities associated with the medications he is taking.

What does the ECG show?





ECG 67 Analysis: Normal sinus rhythm, intraventricular conduction delay, short QT interval, U waves, digoxin effect

There is a regular rhythm at a rate of 68 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is prolonged (0.12 sec) with a normal morphology. Hence this is an intraventricular conduction delay. The axis is physiologically leftward between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF).

The QT/QTc intervals (↔) are very short (280/300 msec), and there is a very prominent U wave (^) seen after the T wave, most obvious in leads V1–V4. In addition the ST segment in leads I, aVL, and V4–V6 are abnormal (↑), with slightly depressed J point and sagging or hammock-like ST-segment changes, particularly in leads I and aVL. The short QT interval is seen with hypercalcemia and is also seen with digoxin, possibly a result of the increased intracellular calcium concentration. The increase in intracellular calcium, which accounts for the positive inotropic effect produced by the drug, is due to the blockade of sodium-potassium ATPase, an increase in intracellular sodium and an increase in sodium-calcium exchange.

The ST-segment changes are suggestive, but not typical for digoxin effect. Digoxin ST segments have a J point that is at baseline with ST-segment depressions that are sagging, scooped, or hammock-like. The ST-segment changes in leads I and aVL, however, are suggestive of digoxin effect. The prominent U waves are seen in hypokalemia. This is likely the result of diuretics and the development of hypokalemia. However, prominent U waves may also be seen with digoxin. The findings on the ECG, *ie*, short QT interval, ST-segment changes, and prominent U wave are all suggestive of therapy with digoxin and probably diuretic use. These changes are representative of a digoxin effect, not digoxin toxicity. The manifestations of digoxin toxicity are the arrhythmias associated with digoxin. ■

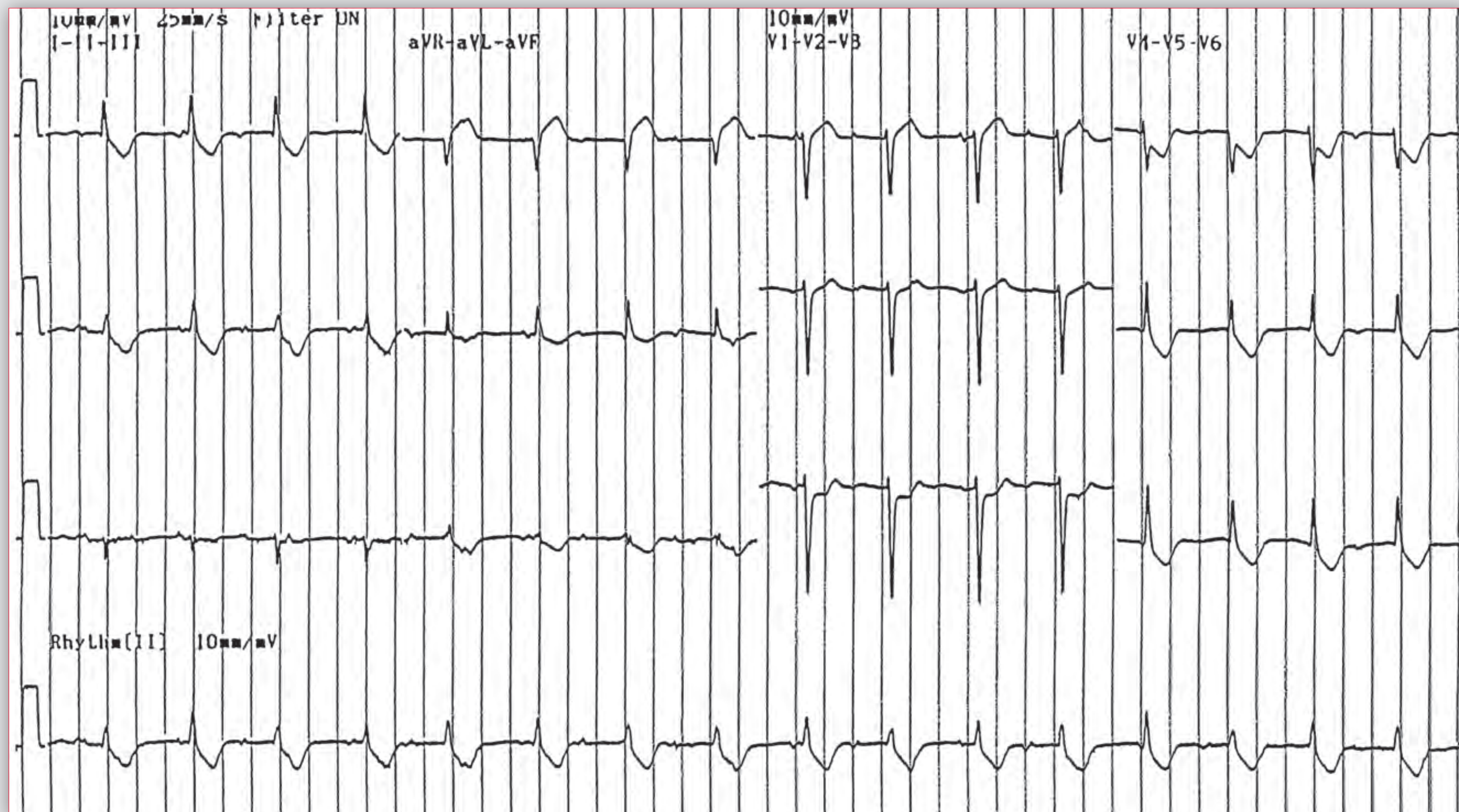
Notes

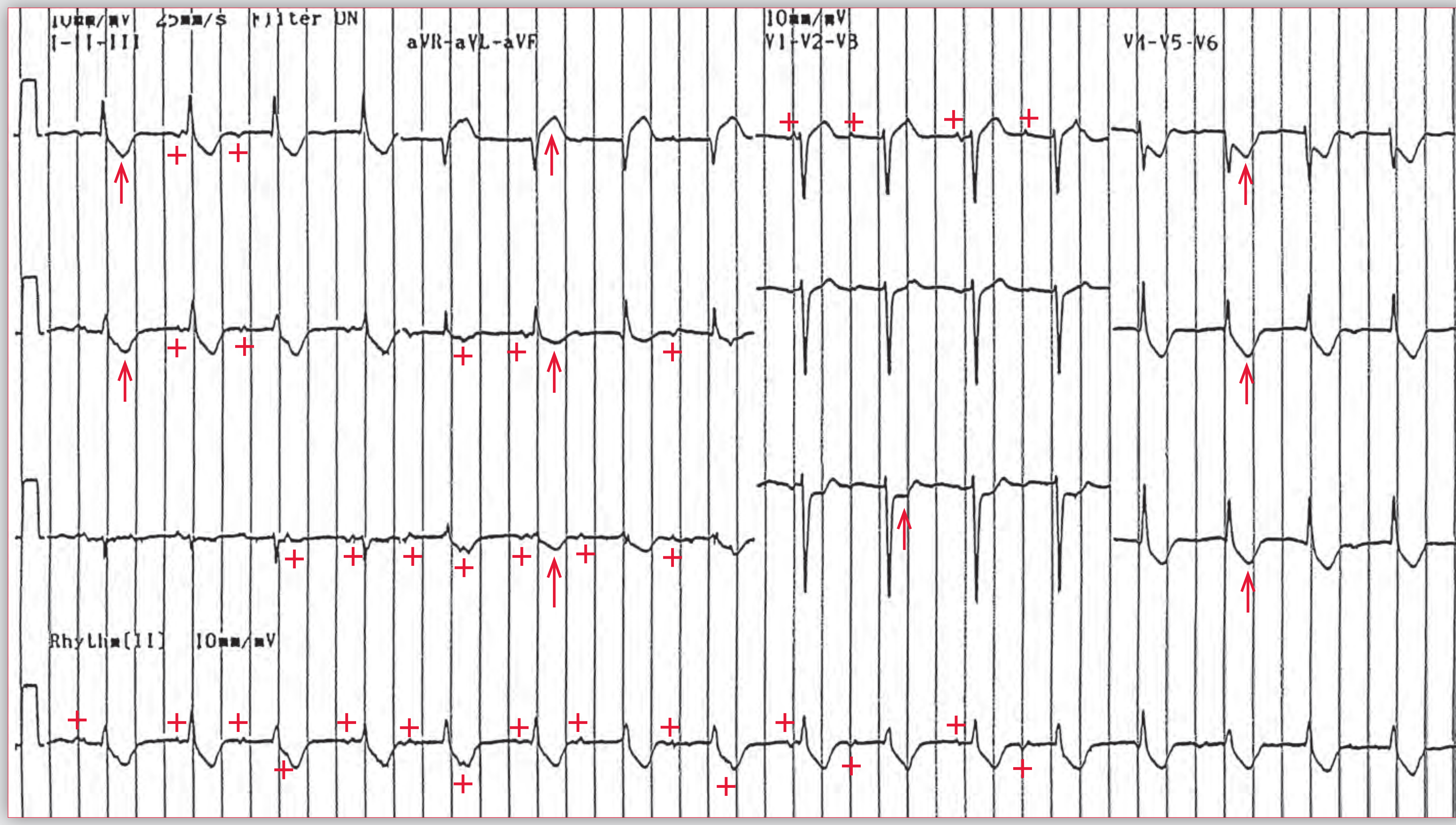
Core Case 68

A 70-year-old woman with a history of paroxysmal supraventricular tachycardia is being treated with digoxin. She presents to her physician with complaints of weakness and nausea and vomiting. An ECG is obtained.

What is the underlying rhythm?

What is the etiology of the abnormality?





ECG 68 Analysis: Ectopic atrial tachycardia, complete heart block, junctional rhythm, ST-T wave abnormalities, digoxin toxicity

There is a regular rhythm with a rate of 100 bpm. The QRS complex duration is normal (0.08 sec). There is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (300/390 msec). There are significant ST-T wave abnormalities (↑) with sagging ST segments as well as J-point depression. The ST changes are very suggestive of a digoxin effect (particularly in leads I, II, aVl, and aVF), although the J-point depression, as seen in leads V4–V6, is not typical.

There are P waves (+) seen, particularly in leads I, II, III, aVL, and aVF. They are small, diminutive, spikey-looking, and have an abnormal morphology, being negative or biphasic (negative positive) in leads I, II, and aVF. The atrial rate is 150 bpm and slightly irregular. Therefore, this is an ectopic atrial tachycardia. There is no relationship between the P waves and the QRS complexes; hence there is AV dissociation with an atrial rate that is faster than the ventricular rate. Therefore, this is an atrial tachycardia with complete heart block and an escape junctional rhythm. This is an arrhythmia that is associated with digoxin toxicity and has often been termed "paroxysmal atrial tachycardia (PAT) with block." The very diminutive P waves that are narrow and have a spike appearance are very typical for digoxin toxicity. This arrhythmia is not life threatening and hence the use of Digibind is not absolutely necessary, although it might be considered.

Digoxin enhances parasympathetic (vagal) tone that results in slowing of sinus node impulse generation and slows conduction through the AV node. Excessive levels of plasma digoxin also result in enhancement of central sympathetic tone and output. In addition, excessive digoxin produces augmentation of delayed afterdepolarizations, which result from an increase an inward flux of calcium. Delayed afterdepolarizations are low-amplitude oscillations of membrane potential that occur after the completion of phase 3 of the action potential, during the early part of phase 4. If the amplitude of these afterdepolarizations is increased, as occurs in the presence of increased sympathetic activity or catecholamines, membrane threshold may be reached, resulting in the occurrence of a spontaneous action potential. This is termed triggered activity, *ie*, electrical activity is triggered by the action potential. Digoxin toxic arrhythmias are the result of enhanced vagal tone, which suppresses normal pacemaker activity, and the development of triggered activity and its augmentation by enhanced sympathetic activity. Thus as the sinus rate slows, there is the development of an ectopic atrial rhythm due to triggered activity. With an increasing level of digoxin, there is an increase in sympathetic output that increases the rate of the atrial rhythm with the development of a tachycardia. As a result of peripheral vagal tone, conduction through the AV node is impaired and blocked, resulting in atrial tachycardia with block. If complete AV block develops there is an atrial tachycardia with an escape junctional rhythm. Increased sympathetic tone will result in an acceleration of the junctional rate, resulting in a junctional tachycardia. ■

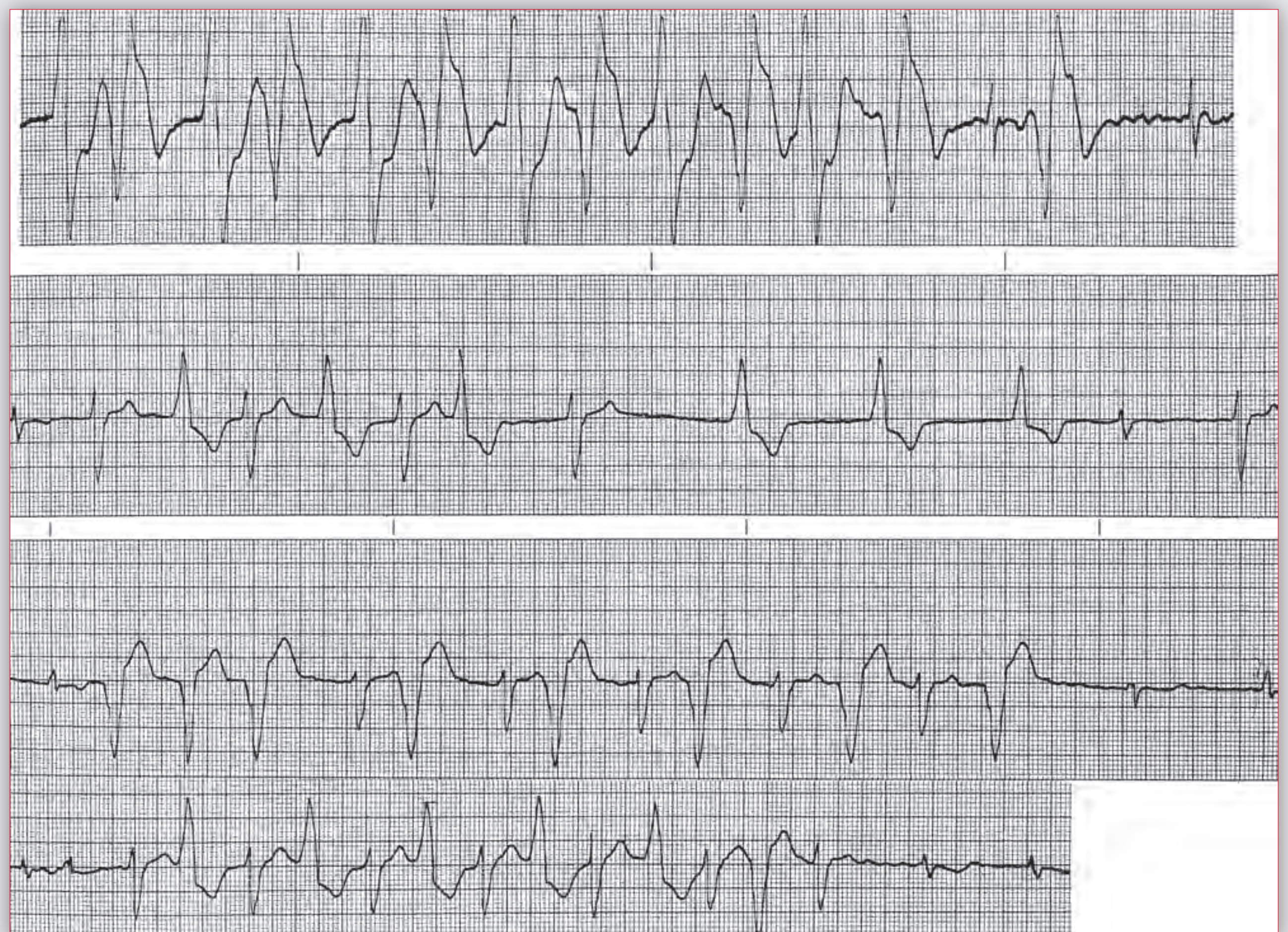
Notes

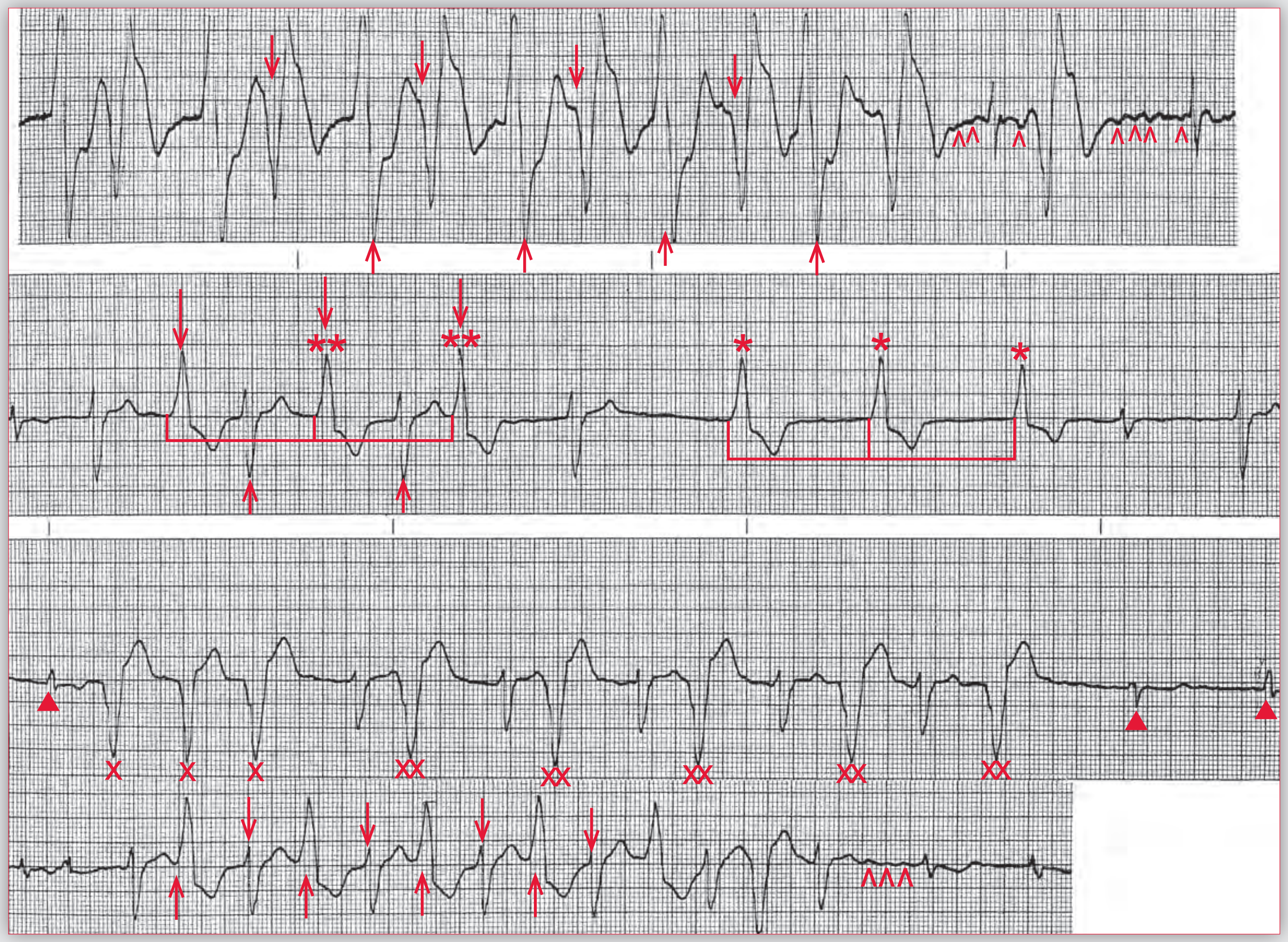
Core Case 69

A 77-year-old man presents to the emergency department with complaints of profound weakness, nausea, and vomiting. He has been unable to eat or drink much for the past 4–5 days because of these GI complaints. He has a history of heart failure for which he is receiving digoxin and diuretics. While on telemetric monitoring, a change in rhythm is noted.

What arrhythmia is noted?

What is the etiology for this arrhythmia?





ECG 69 Analysis: Atrial fibrillation, bidirectional tachycardia, premature ventricular complexes (ventricular bigeminy), junctional rhythm, digoxin toxicity

There are several rhythm strips seen from different telemetry leads. The underlying rhythm is atrial fibrillation, as can be seen at the end of the first rhythm strip with the presence of irregular and rapid undulations (^) of the baseline. The first rhythm strip shows a regular rhythm at a rate of 130 bpm and there are beat-to-beat changes in QRS morphology (↑, ↓). The same pattern of beat-to-beat changes in QRS morphology (↑, ↓) can be seen in the second and fourth rhythm strips. This is termed bidirectional junctional tachycardia (due to alternating right [RBBB] and left bundle branch block [LBBB]).

The second rhythm strip shows episodes of regular QRS complexes (*) at a rate of 50 bpm. These are junctional complexes and they have the same morphology as one of the QRS complexes (**) during the preceding tachycardia. In addition, the interval between these QRS complexes (*ie*, rate 50 bpm) (⊔) is the same as the RR interval (⊔) of the QRS complexes with the same morphology (**) that are seen during the tachycardia The third rhythm strip shows 3 sequential QRS complexes (x) that have a morphology and width that is different from the supraventricular complexes (▲) (complex number 1 and the last two). This is nonsustained ventricular tachycardia. In addition there are premature ventricular complexes (xx) in a bigeminal pattern that have the same morphology as the QRS complexes of the nonsustained ventricular tachycardia.

In the presence of digoxin, there is an increase in vagal tone and hence a slowing of the ventricular rate during atrial fibrillation. The earliest manifestation of excessive digoxin is intermittent long RR intervals, each one is the same in duration; this represents intermittent complete heart block. With an increase in digoxin levels, there is persistent complete heart block and regularization of the RR interval. This represents complete heart block with an escape junctional rhythm. As the digoxin level increases further, the junctional rate increases, resulting in a nonparoxysmal junctional tachycardia. As the digoxin level increases still further, there is conduction block within the His-Purkinje system, resulting in intermittent RBBB and LBBB, occurring in an alternating pattern. This is termed bidirectional tachycardia. This may be a junctional or a bundle of His rhythm with alternating conduction via the right and left bundles. This has also been called a bidirectional ventricular tachycardia, but in the presence of digoxin it is most likely a junctional tachycardia and not ventricular. In order to have alternating RBBB and LBBB, the impulse must come through the bundle of His and hence originate in the junction. As this is a serious digoxin toxic rhythm, the use of Digibind is indicated for therapy.

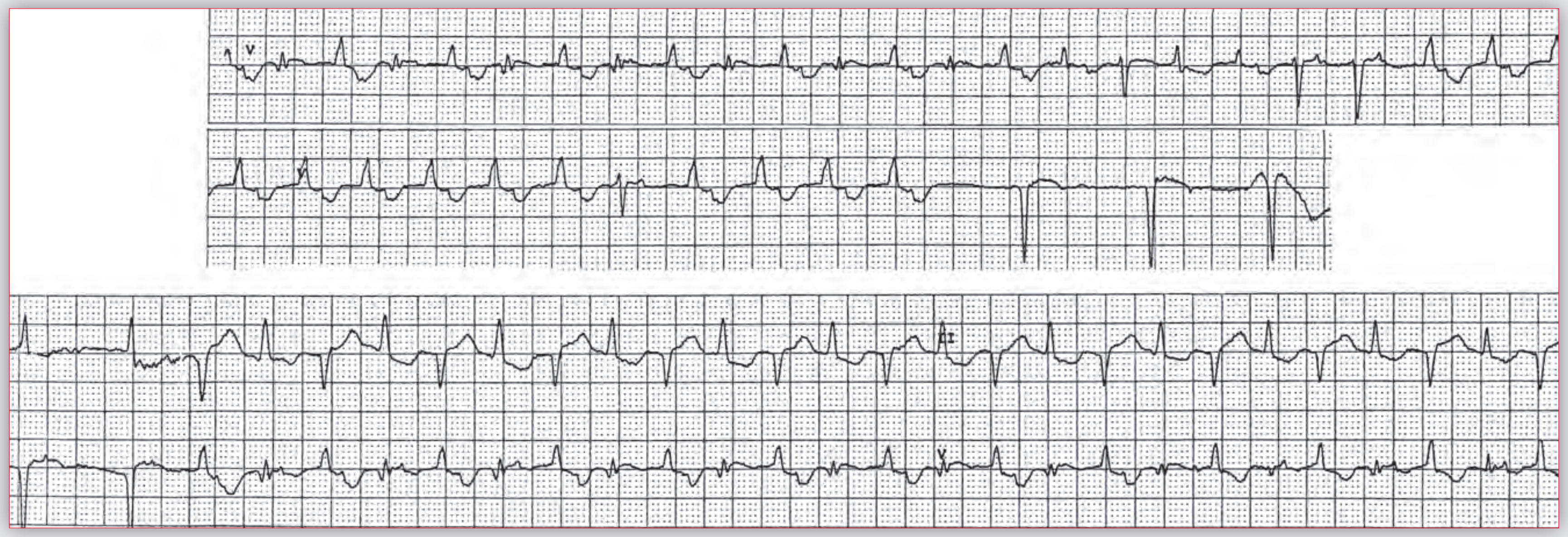
Although bidirectional tachycardia is seen with digoxin toxicity, it may also be seen in other conditions such as severe underlying cardiomyopathy. There is also ventricular tachycardia that demonstrates beat-to-beat changes in QRS complex morphology, *ie*, a bidirectional ventricular tachycardia that is seen with a dilated cardiomyopathy and also in fulminant myocarditis. ■

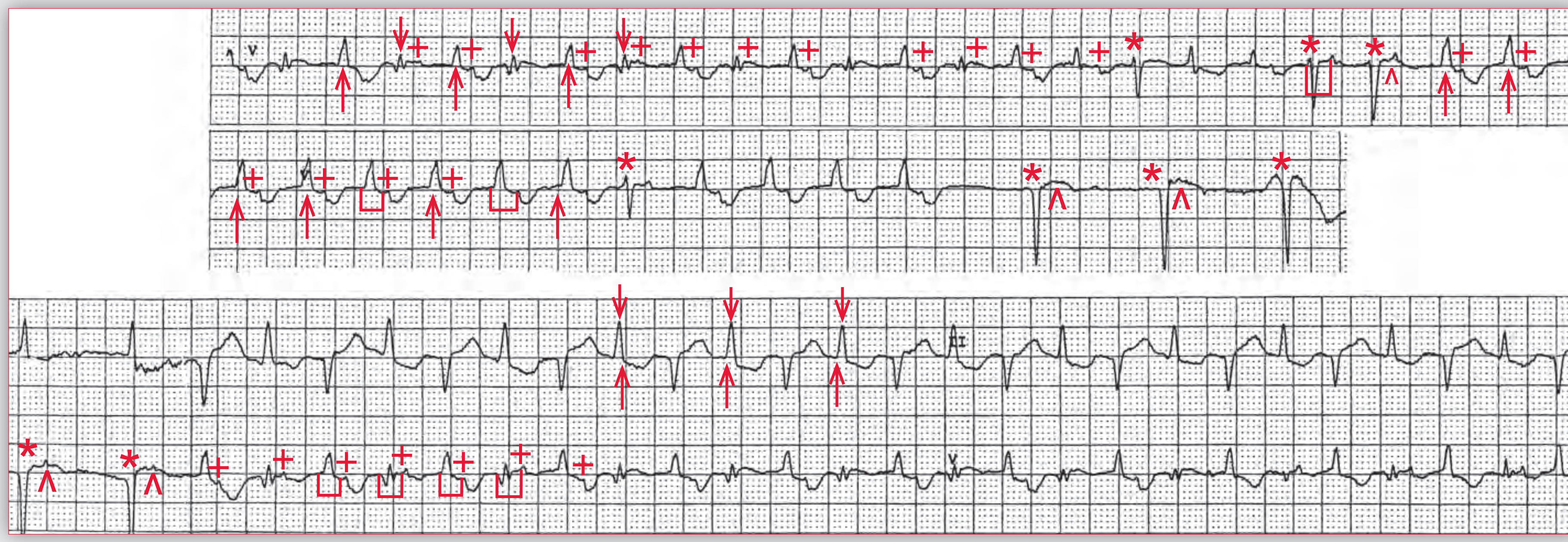
Notes

Core Case 70

A 65-year-old man with a history of atrioventricular nodal reentrant tachycardia (AVNRT) is being treated with digoxin and a β-blocker. As a result of an increase in palpitations, his physician suggests that he double the dose of the β-blocker. However, the patient misunderstood these directions, and in error he doubled the dose of digoxin. One week later, he came to the emergency department with complaints of abdominal pain and nausea. He was placed on telemetry.

What rhythm is seen on the telemetry strips?





ECG 70 Analysis: Bidirectional junctional tachycardia

The first two rhythm strips (lead V1) are continuous, while the bottom rhythm strip shows two simultaneously recorded leads (II and V1). There is a regular rhythm at a rate of 156 bpm. The QRS complex duration is increased (0.12 sec), although several narrow QRS complexes are noted (*). There are retrograde P waves (+) seen after each QRS complex, including the narrow QRS complexes (^). The RP interval (⊔) is stable (0.16 sec). Hence this is a junctional tachycardia. The first two rhythm strips show episodes of alternating conduction patterns (↑, ↓) (right [RBBB] and left bundle branch block [LBBB] morphologies) followed by QRS complexes, all of which have a RBBB morphology (↑) that is the same as the RBBB morphology seen during the initial portion of the rhythm strip when there is alternating conduction patterns. Seen in the bottom rhythm strip are QRS complexes with alternating conduction patterns (↑, ↓) (RBBB and LBBB). Therefore, this is a bidirectional junctional tachycardia, which is a classic digoxin toxic arrhythmia. This patient presents with typical clinical symptoms of digoxin toxicity (abdominal pain and nausea), due to having taken twice the normal dose.

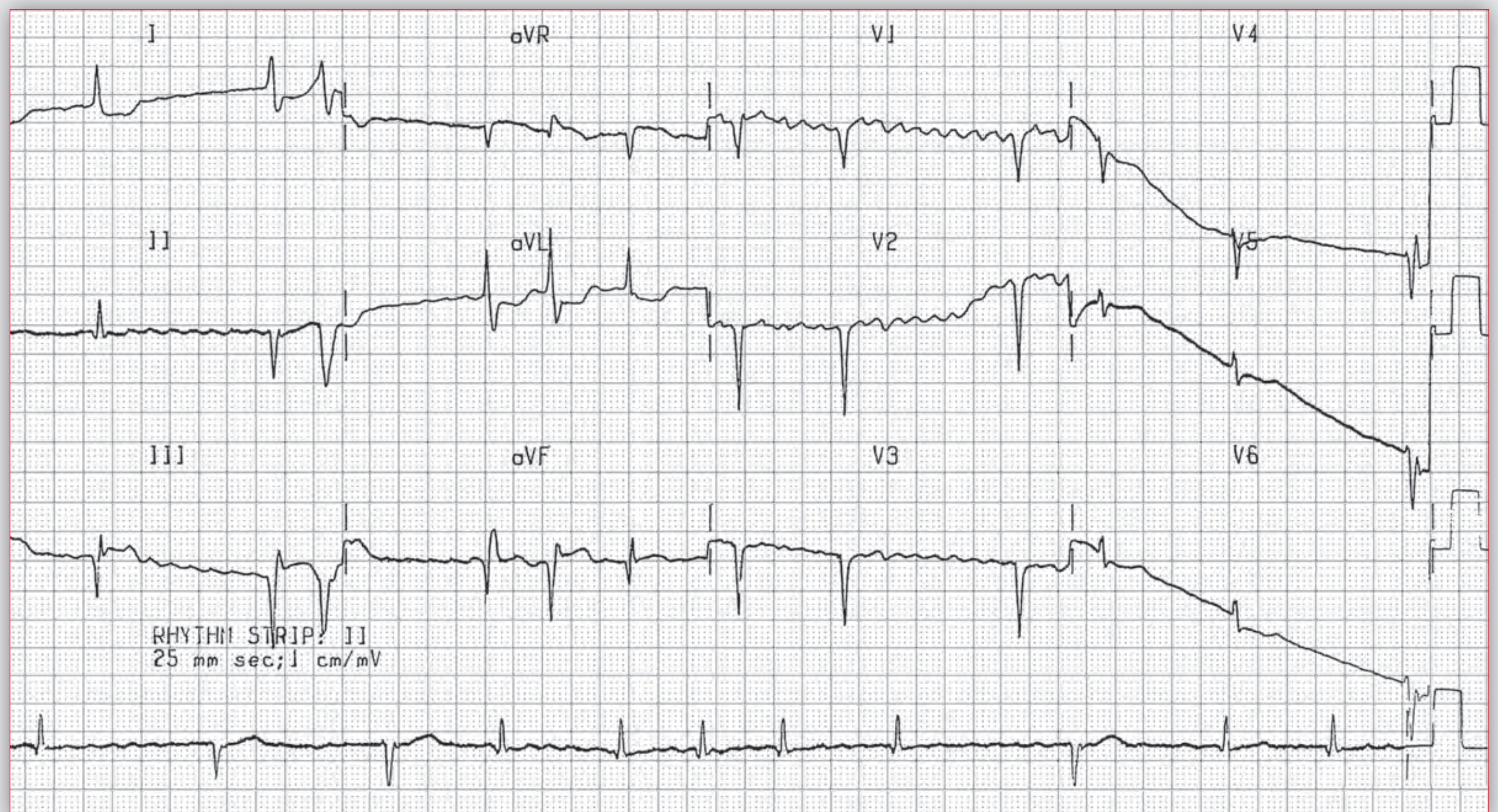
Digoxin enhances parasympathetic (vagal) tone that results in slowing of sinus node impulse generation and slows conduction through the AV node. Excessive levels of plasma digoxin also result in enhancement of central sympathetic tone and output. With digoxin toxicity, there is suppression of sinus node automaticity (due to enhanced vagal tone) with an increase in automaticity of lower foci—in this case, the AV node—with the development of an ectopic junctional tachycardia with intact VA conduction. As the digoxin level increases further, there is conduction block within the His-Purkinje system, resulting in intermittent RBBB and LBBB, occurring in an alternating pattern. There may also be alternating left anterior and left posterior fascicular block. This is termed bidirectional tachycardia. This may be a junctional or a bundle of His rhythm with alternating conduction via the right and left bundles. This has also been called a bidirectional ventricular tachycardia, but in the presence of digoxin, it is a junctional tachycardia.

Although bidirectional tachycardia is seen with digoxin toxicity, it may also be seen in other conditions such as severe underlying cardiomyopathy. There is also ventricular tachycardia that demonstrates beat-to-beat changes in QRS complex morphology, *ie*, a bidirectional ventricular tachycardia that may be seen with a dilated cardiomyopathy or a fulminant myocarditis. ■

Core Case 71

A 66-year-old woman with a history of chronic obstructive pulmonary disease (COPD) and atrial fibrillation, for which she is receiving digoxin for ventricular rate control, presents to the emergency department with complaints of nausea, vomiting, and abdominal pain. She states that for the past 2 weeks she has been receiving erythromycin, which was prescribed for a COPD exacerbation. Since she started the drug, she has noted anorexia and has been not eating or drinking much. Over the past 2 days she has noted nausea and vomiting and this morning awoke with

ECG 71A



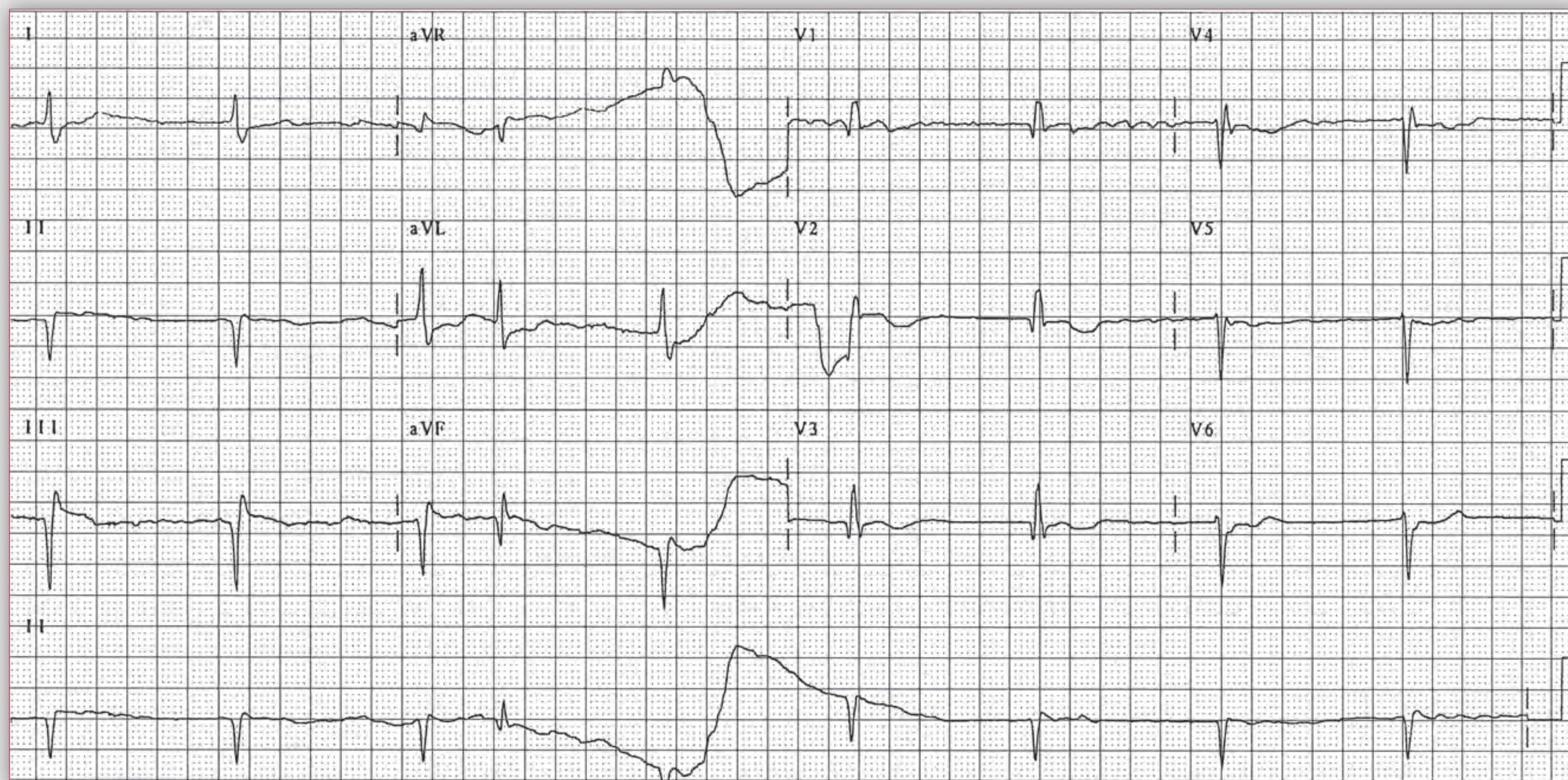
abdominal pain. Her physical examination is remarkable for dry mouth and poor skin turgor, as well as other signs of dehydration. Her laboratory tests demonstrate a BUN that is elevated to 76 mg/dL with a creatinine of 2.5 mg/dL, both of which are higher than her baseline values, which are normal. An ECG (ECG 71A) is obtained. She is begun on IV hydration and there is an improvement in her BUN and creatinine. However, on the following day she is still complaining of abdominal pain and severe lethargy. Another ECG is obtained (ECG 71B).

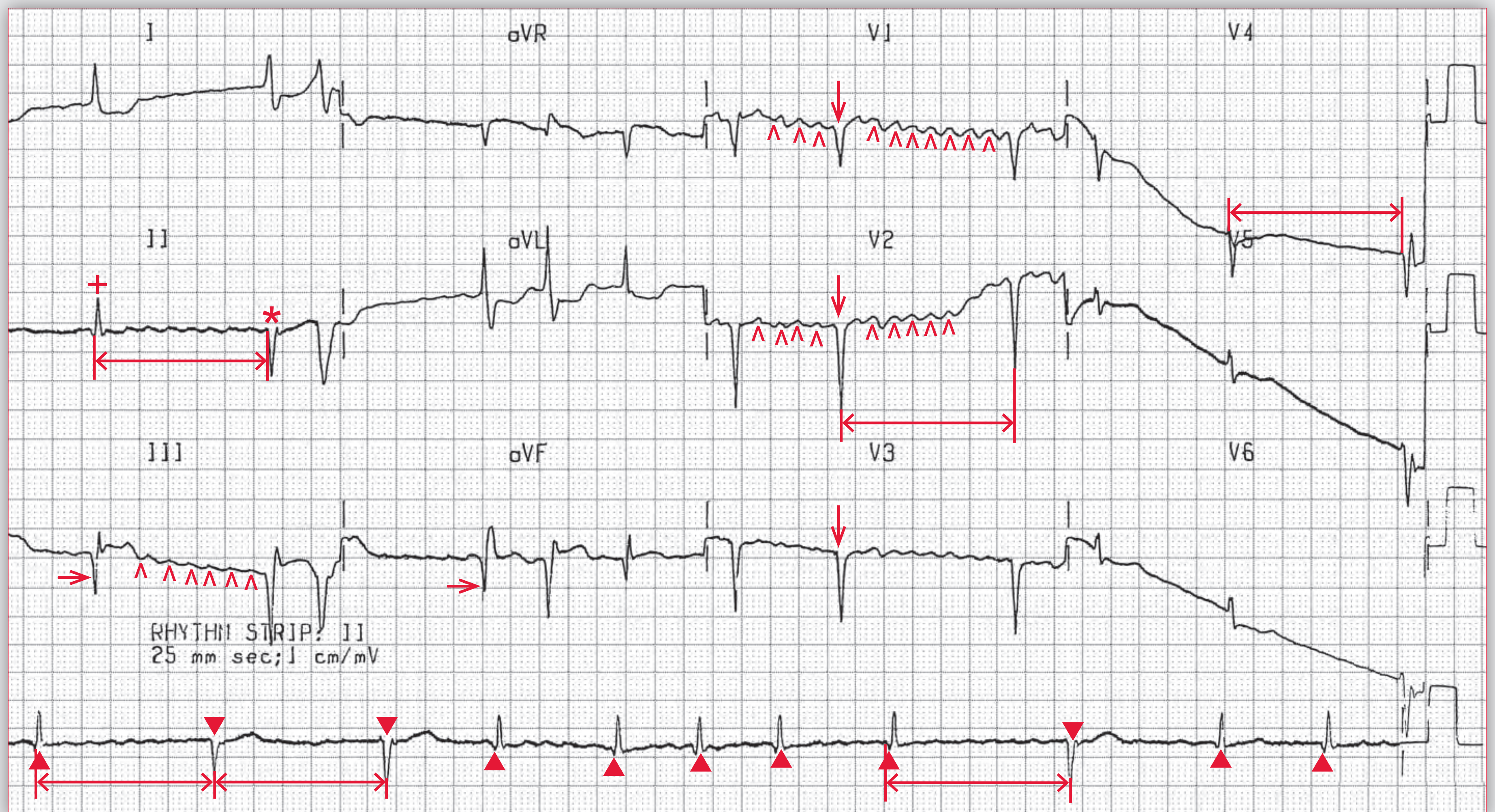
What does this ECG show?

What further testing would be indicated?

What is the possible cause for the ECG changes?

ECG 71B



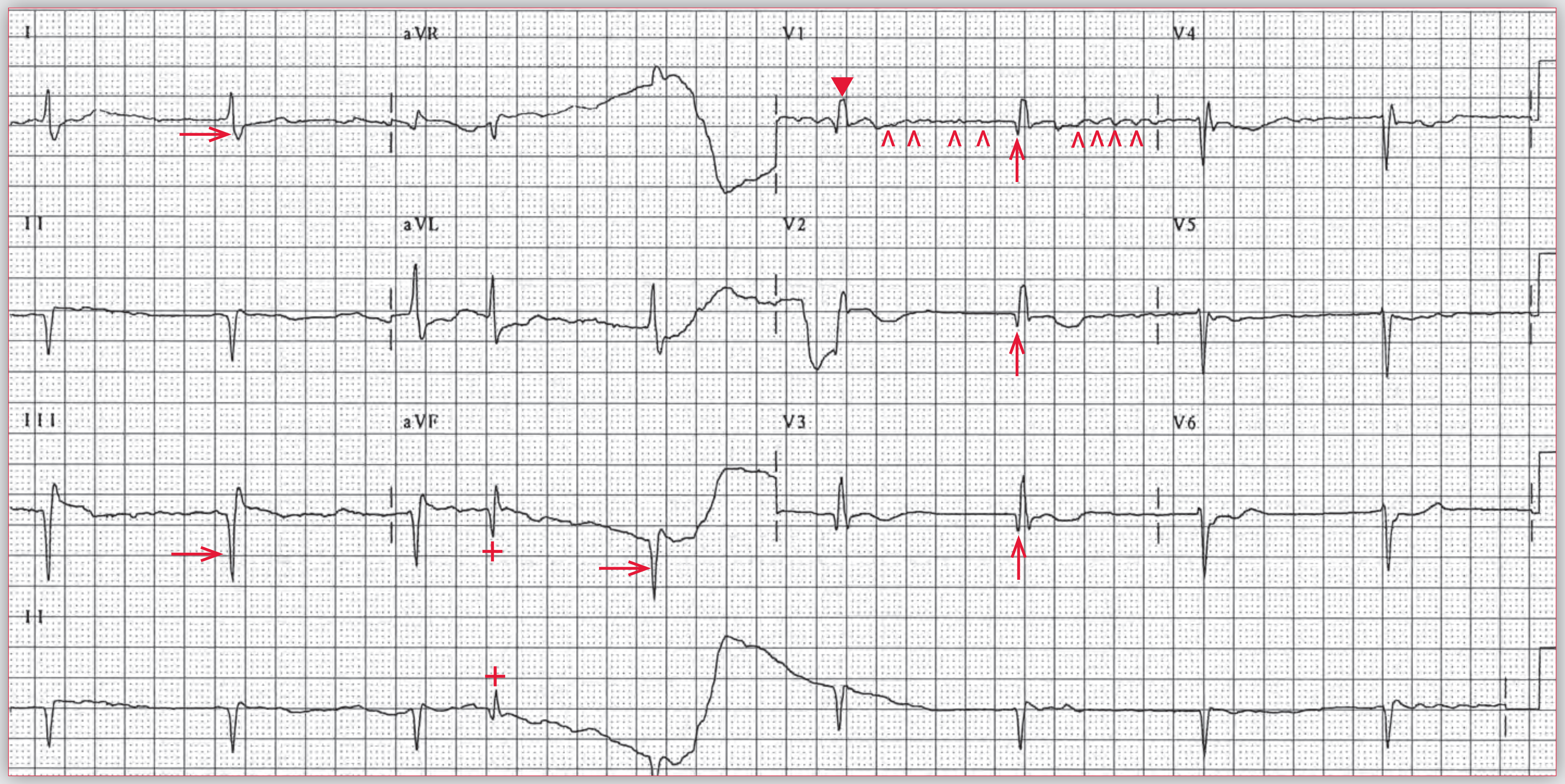


ECG 71A Analysis: Atrial fibrillation, intermittent regularization due to complete heart block with an escape junctional rhythm, old anteroseptal myocardial infarction, old inferior wall myocardial infarction.

In **ECG 71A**, it should be noted that the rhythm strip is not simultaneous with the 12-lead ECG recording. The rhythm is irregularly irregular with an average rate of 66 bpm. There are no obvious P waves seen. However, there are rapid and irregular undulations of the baseline (^), most obviously seen in leads II, III, and V1–V3. Hence the rhythm is atrial fibrillation. The QRS complex duration is normal (0.08 sec). There are no R waves in leads V1–V3 (↓), consistent with an old anteroseptal myocardial infarction. In addition, there are significant Q waves in leads III and aVF (→), suggesting an old inferior wall myocardial infarction. Although the QRS complex morphology is consistent, there are changes in the QRS complex axis. All the QRS complexes are positive in lead I and negative in lead aVF. However, it can be seen in lead II that the first QRS complex (+) is positive (and hence associated with a leftward axis between 0° and –30°), while the second QRS complex (*) is negative (and hence associated with a more extreme left axis between –30° and –90°). This can also be seen on the lead II rhythm strip; the first, fourth through eighth, tenth, and eleventh complexes (▲) have a leftward axis, while the second, third, and ninth complexes (▼) have a more extreme left axis. Also noted is that the complexes with a more extreme left axis always follow a long pause (RR interval) with a rate of 60 bpm, and each time this RR interval is the same (↔). Thus every long RR interval has the same duration (1.20 sec). Therefore, this is intermittent regularization during atrial fibrillation and is characteristic of intermittent complete AV block with an escape QRS complex that always has the same escape interval. The QRS complex following the pause has the same duration and morphology as the conducted QRS complex, the only difference being an axis shift. Hence the escape rhythm is junctional. Junctional complexes commonly have a slightly different amplitude or axis compared to complexes due to conduction through the AV node. This is likely because the impulse originating from an ectopic junctional focus enters the bundle of His at a different location and is conducted through the His-Purkinje tissue through a different tract compared to impulses coming through the AV node.

In this patient, who appears to be dehydrated as a result of poor oral intake and who is being treated with erythromycin (an antibiotic reported to increase the level of digoxin), the ECG is very suggestive of early digoxin toxicity, with the occurrence of intermittent complete heart block, noted as intermittent regularization of the RR intervals. Further supporting digoxin toxicity are the symptoms of nausea, vomiting, abdominal pain, and lethargy. Although these are symptoms that can occur with many conditions and are nonspecific, they are frequently seen with digoxin toxicity.

continues



ECG 71B Analysis: Atrial fibrillation with regularization due to complete heart block and an escape junctional rhythm, right bundle branch block, old inferior wall myocardial infarction, old anterior wall myocardial infarction.

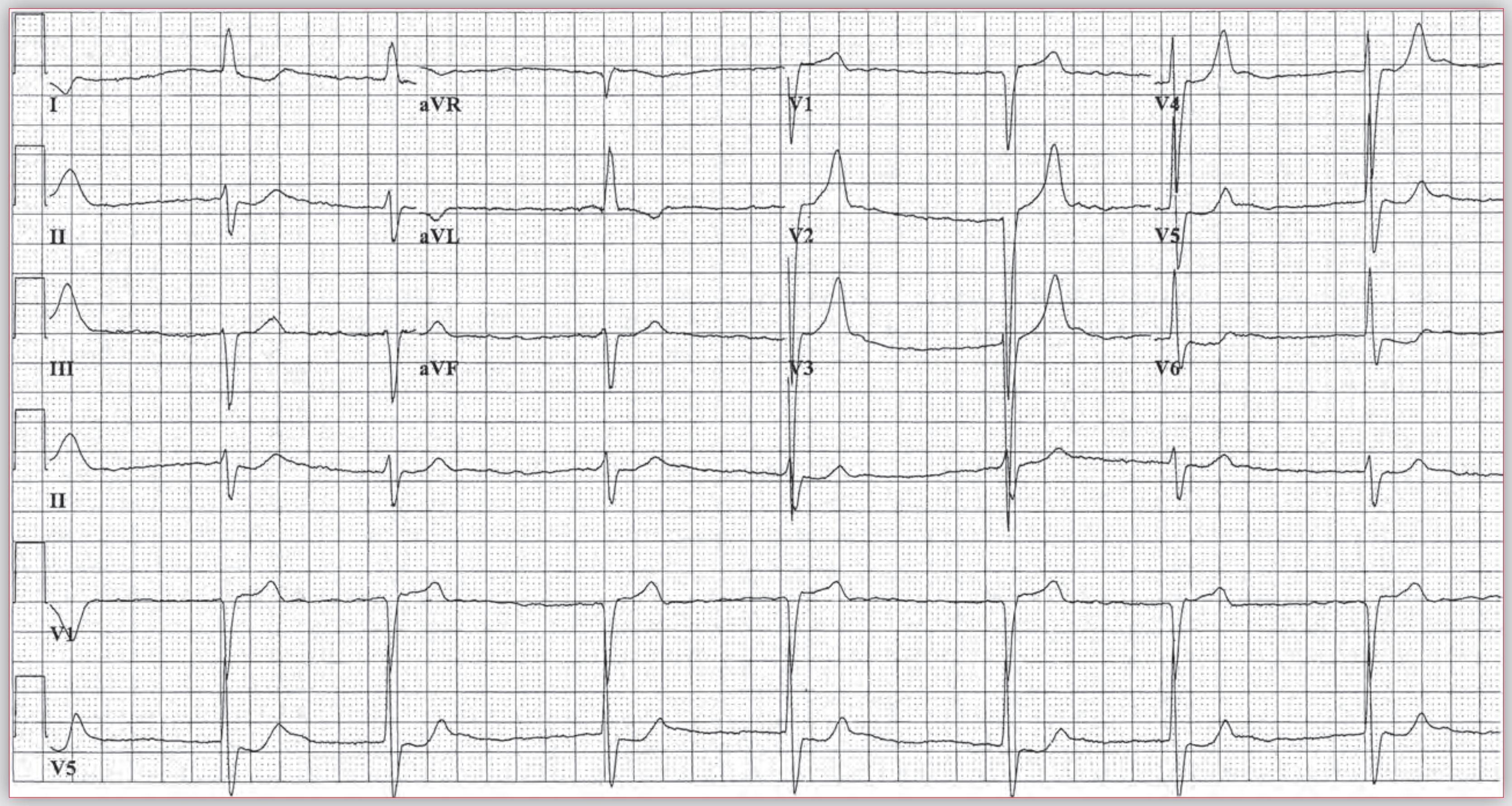
On the following day, **ECG 71B** is obtained, and this shows a regular rhythm at a rate of 60 bpm (RR intervals 1.2 sec). On this ECG the rhythm strip is simultaneous with the 12-lead ECG. The RR intervals and rate are identical to what was seen with the long RR intervals on ECG 71A. The rhythm is still atrial fibrillation with fine undulations of the baseline (^) seen in leads II, III, aVF, and V1). Noted is that the QRS complex morphology is similar to that on ECG 71A, and there is an extremely leftward axis, which is identical to the axis of the escape junctional complex on ECG 71A. Hence this is complete heart block with an escape junctional rhythm. The QRS complex have inferior Q waves (→) as well as initial Q waves in leads V1–V3 (↑), consistent with the previous diagnosis of an inferior and anteroseptal myocardial infarction. However, the complex is now wider (0.12 sec) and there is a morphology of a right bundle branch block with a broad S wave in lead I (←) and a broad R wave in V1 (▼). It should also be noted that the fourth QRS (**+**) is early and has a morphology and axis that are identical to the conducted complexes on ECG 70A. Hence there is intermittent AV conduction noted.

The progression from intermittent to more consistent complete (third-degree) AV block is suggestive of a further increase in the digoxin level and is now certainly diagnostic of an excess digoxin effect or toxicity. This could be confirmed with check a digoxin level and certainly discontinuing the digoxin at this time. As the rhythm abnormality is not serious or life threatening, this is not clinical indication for the use of Digibind. ■

Core Case 72

A 68-year-old man with chronic atrial fibrillation at baseline(ECG 72A) treated with a β-blocker and digoxin, is seen in the office for a routine follow up. He denies any symptoms. An ECG is obtained (ECG 72B) and is noted to be different from his baseline ECG (ECG 72A).

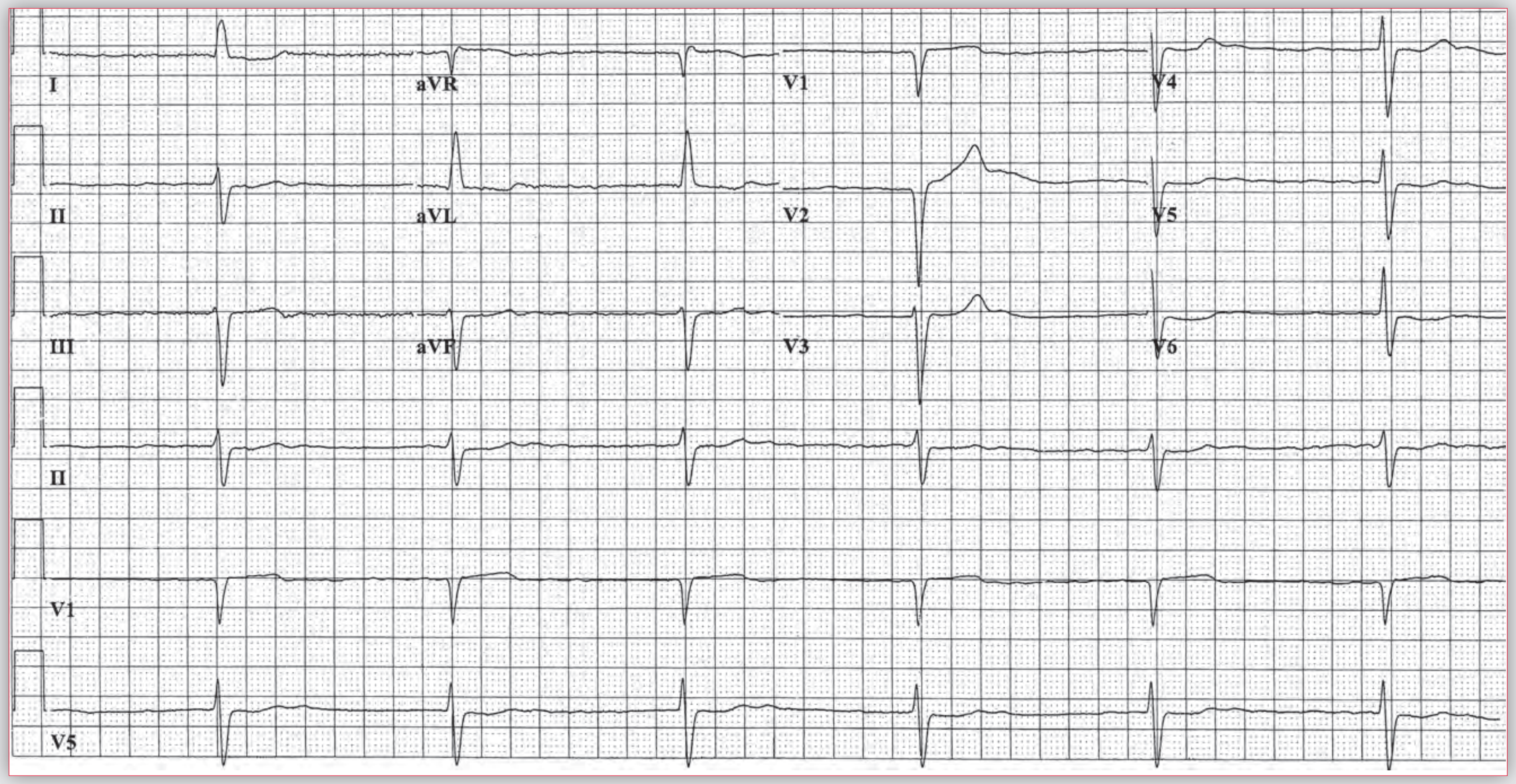
ECG 72A

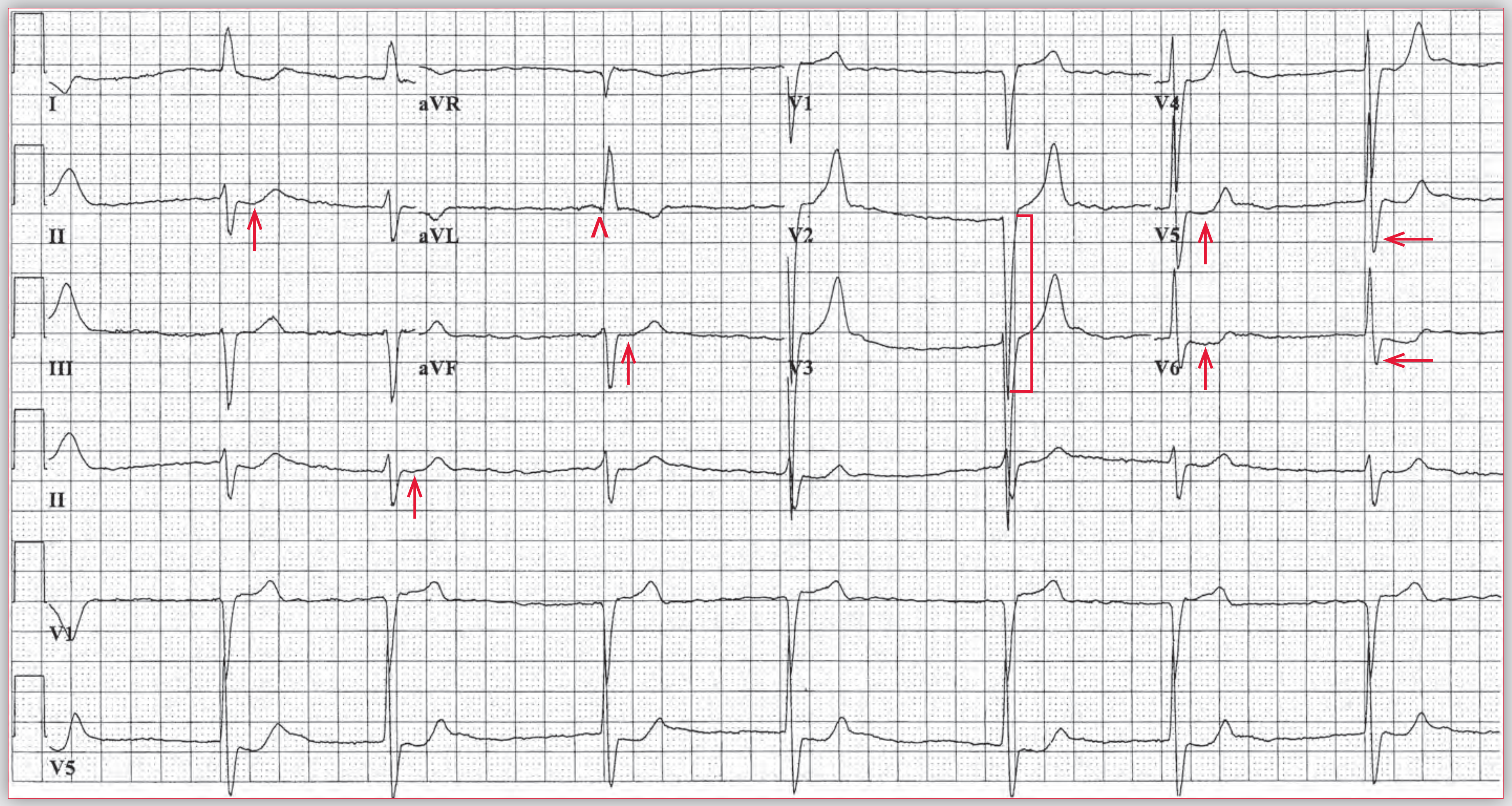


What is the abnormality noted on ECG 72B?

What are the potential causes for the finding?

ECG 72B

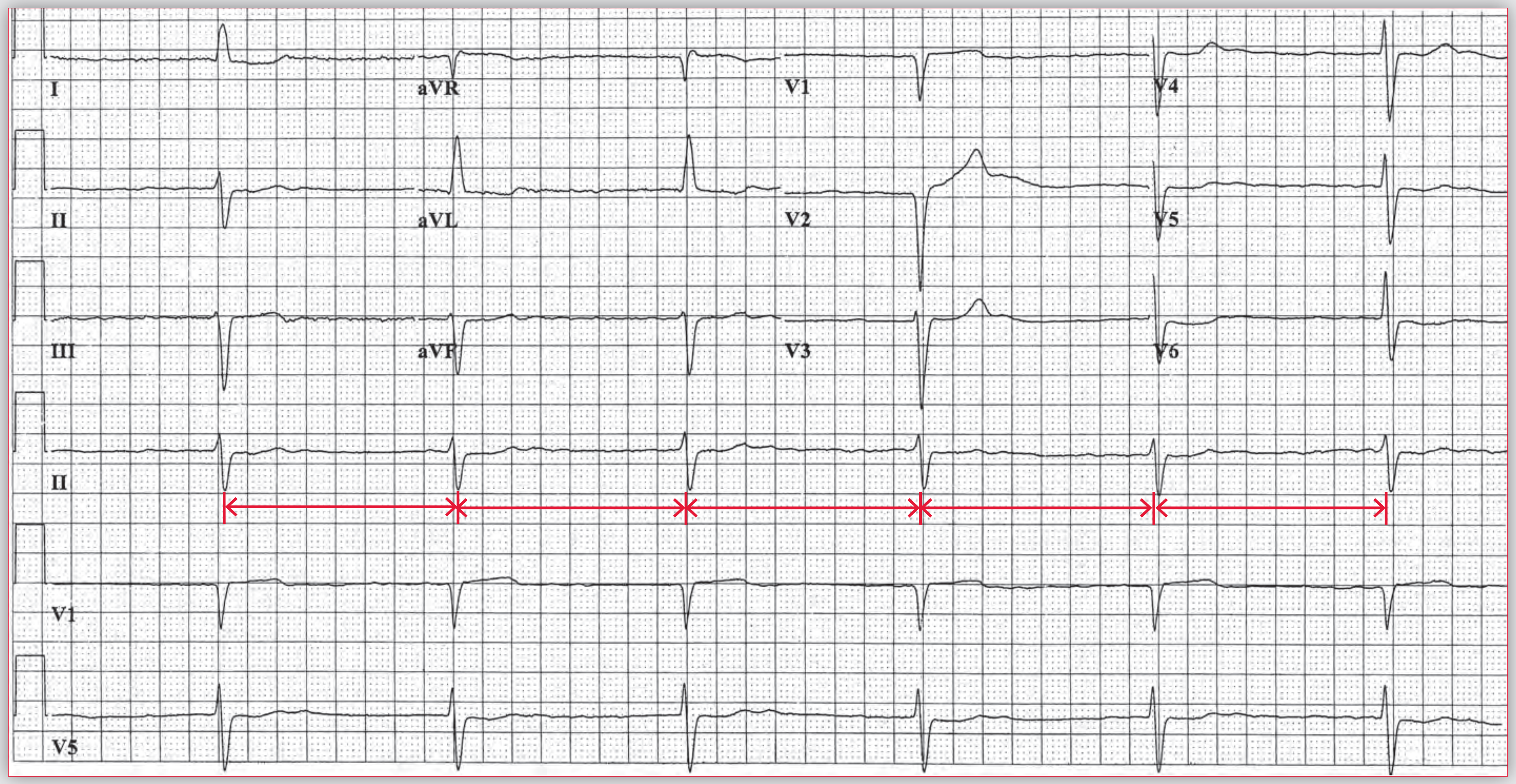




ECG 72A Analysis: Atrial fibrillation, intraventricular conduction delay (IVCD), left anterior fascicular block (LAFB), left ventricular hypertrophy, ST-T wave changes (possible digoxin effect)

ECG 72A is the baseline ECG from the patient and can be compared to ECG 72B recently obtained. The rhythm is irregularly irregular at an average rate of 44 bpm. There are no P waves seen before or after the QRS complexes, although fine undulations of the baseline are seen. The rhythm is atrial fibrillation with a slow ventricular response. The QRS complex duration is prolonged (0.12 sec), but there is no pattern for a right or left bundle branch block. Noted is a Q wave in lead aVL (^) as well as terminal S waves in leads V5–V6 (←). Hence this is an intraventricular conduction delay. There is an extreme leftward axis between –30° and –90° (positive QRS complex in lead I and negative in leads II and aVF with an rS morphology). Hence this is a LAFB. The QT/QTc intervals are normal (460/410 msec and 440/380 msec). The S wave in V2 is 30 mm ( ]), suggesting the presence of left ventricular hypertrophy (*ie*, R or S wave in any precordial lead ≥ 25 mm). There are QS complexes in leads V1–V2 (↓), which is seen with a LAFB, although is also suggestive of an anteroseptal myocardial infarction. There are ST-T wave abnormalities in leads I, II, aVF, and V5–V6 (↑), which are consistent with a digoxin effect, *ie*, the J point is at baseline (T-P segment) while the ST segments are sagging, scooped, or hammock-like.

continues



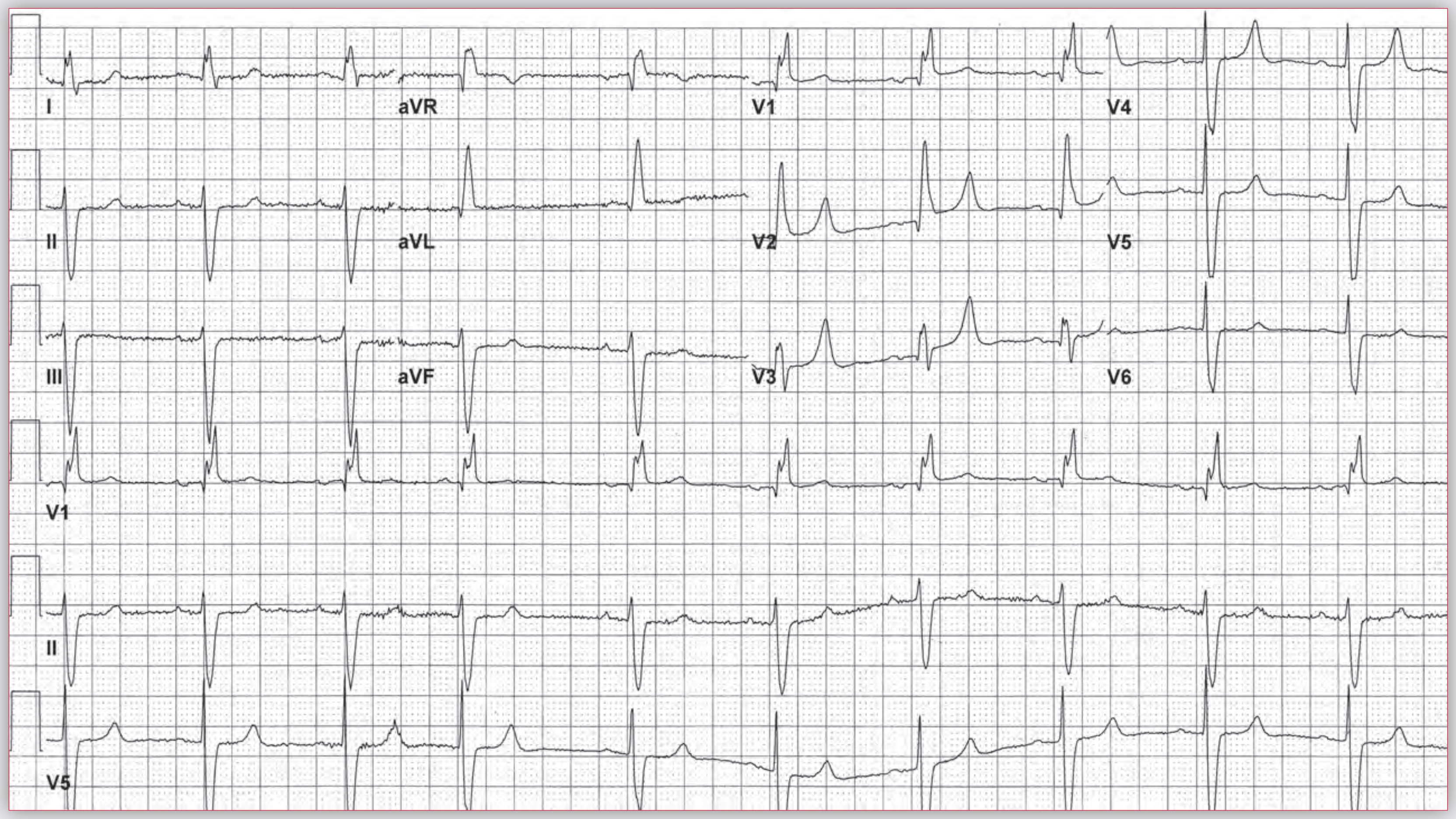
ECG 72B Analysis: Atrial fibrillation, complete heart block, escape junctional rhythm, IVCD, LAFB, left ventricular hypertrophy, ST-T wave changes

ECG 72B: No P waves are seen before or after any QRS complexes, and the rhythm is still atrial fibrillation. The QRS duration, axis, and morphology is the same as in ECG 72A. The QT/QTc intervals are the same as ECG 72A. The ST-segment changes, suggesting a digoxin effect, are still present. However, there is a regular rhythm (↔) at a rate of 38 bpm. Atrial fibrillation is associated with a rhythm and RR intervals that are irregularly irregular. The ventricular rate is dependent upon AV nodal conduction. In this case, the ventricular rate is regular, *ie*, regularized atrial fibrillation. This indicates the presence of complete heart block. As the QRS complexes are identical to those in ECG 72A, this is an escape junctional rhythm. The complete heart block is likely the result of an excessive effect from the AV nodal blocking agents being taken, *ie*, a β-blocker and digoxin. However, the presence of ST-segment changes due to digoxin is a digoxin effect and not digoxin toxicity. The complete heart block may also be the result of intrinsic AV nodal disease or high vagal tone. The appropriate therapy would be to discontinue the digoxin and β-blocker to see if there is resolution of the complete heart block. If so, a reassessment of therapy for rate control would be important. Persistence of complete heart block would mean that there are likely structural problems with the AV node. There should be consideration for a pacemaker. ■

Core Case 73

An 88-year-old man with is admitted with fatigue. He has a history of paroxysmal atrial fibrillation and has been treated with a β-blocker and diltiazem. An ECG is obtained (ECG 73B) while his baseline ECG (ECG 73A) is shown for comparison.

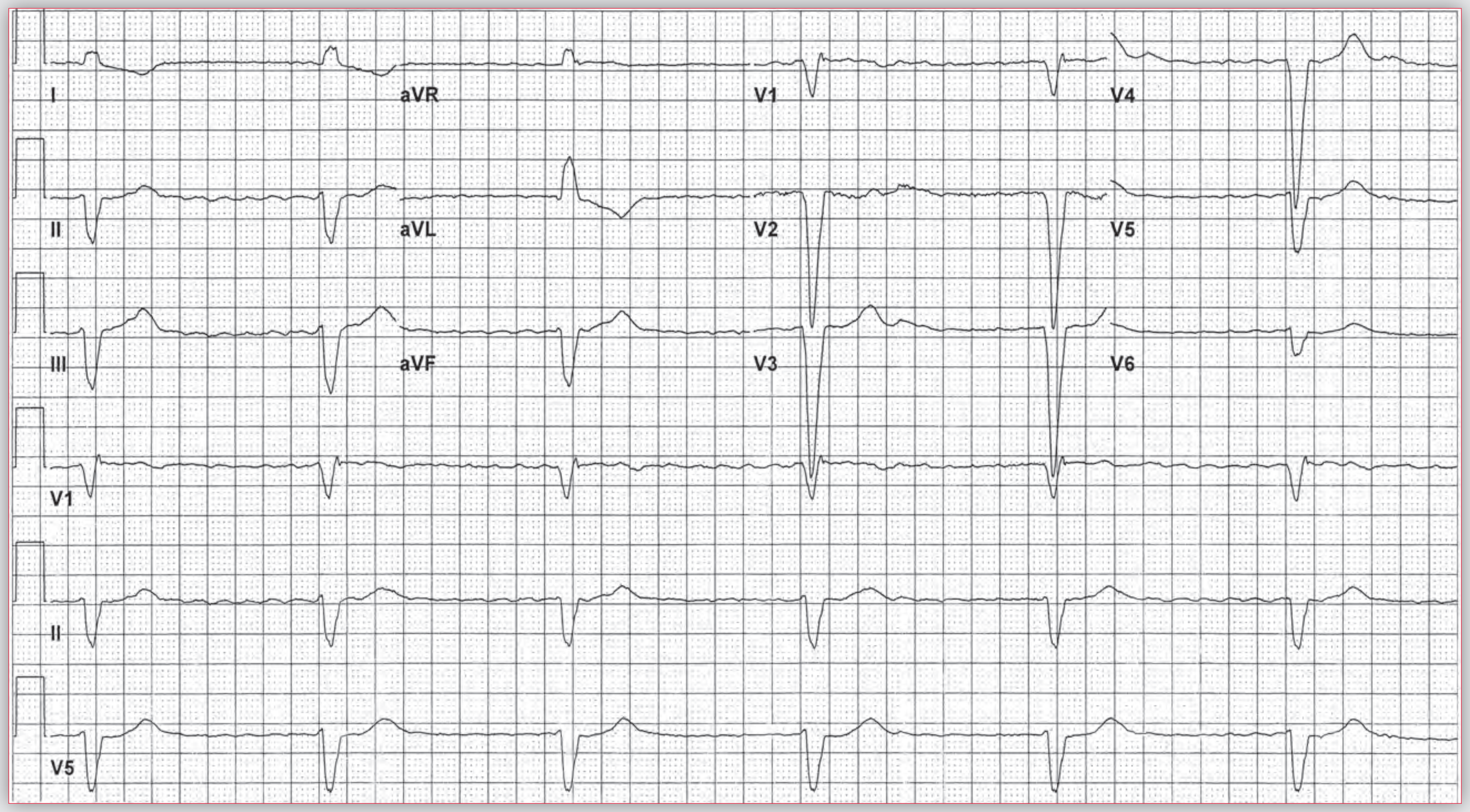
ECG 73A

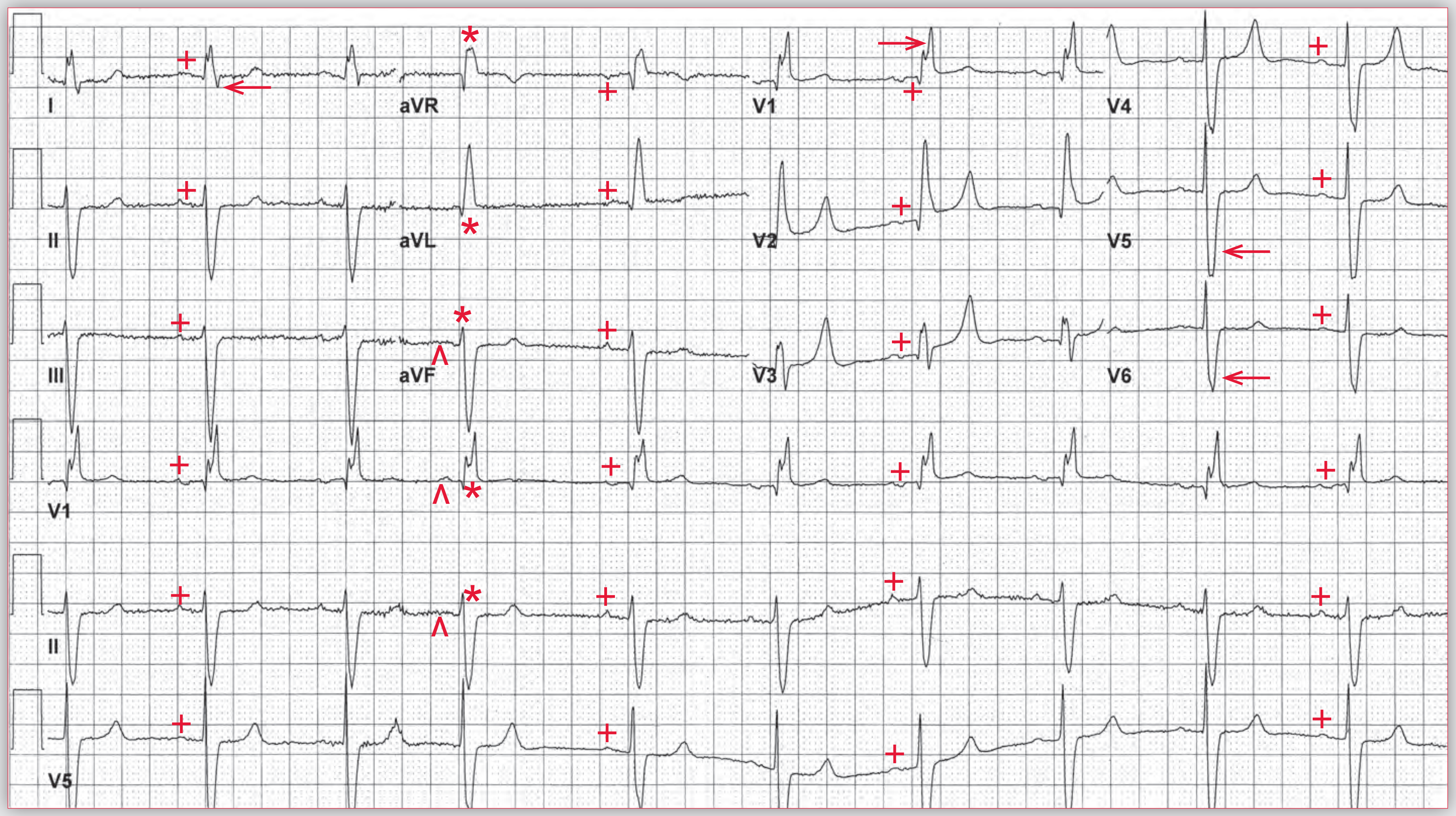


What do these ECGs show?

What is the etiology of his fatigue?

ECG 73B



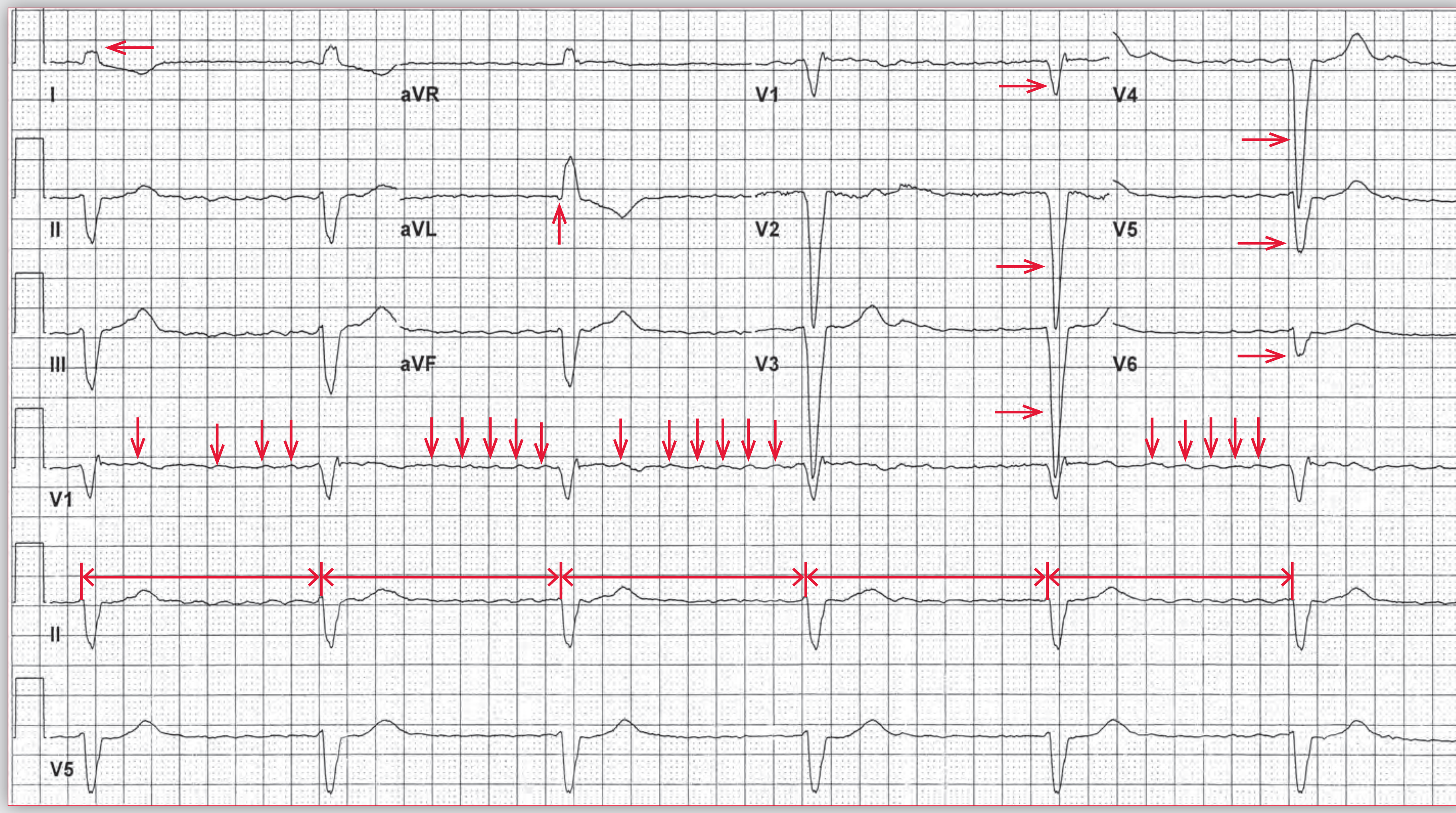


ECG 73A Analysis: Normal sinus rhythm, single premature atrial premature complex, right bundle branch block, left anterior fascicular block (bifascicular block)

ECG 73A shows the rhythm is regular at a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm. The fourth QRS complex is early (*) and has the same QRS morphology as the other QRS complexes. There is a P wave (^) before this complex with a different P wave morphology and shorter PR interval (0.16 sec). Hence this is a premature atrial premature complex.

The QRS complex duration is prolonged (0.14 sec) and there is a right bundle branch block (RBBB) morphology with an RR′ in V1 (→) and broad S wave in leads I and V5–V6 (←). The axis is extremely leftward between −30° and −90° (positive QRS complex in lead I and negative in leads II and aVF with an rS morphology). This is a left anterior fascicular block (LAFB). The presence of a RBBB and LAFB is termed bifascicular block. The QT/QTc intervals are prolonged (480/480 msec) but are normal when the prolonged QRS complex duration is considered (440/440 msec).

continues



ECG 73B Analysis: Atrial fibrillation with complete heart block and an escape ventricular rhythm

ECG 73B is from the same patient as ECG 73A. The rhythm is regular at a rate of 36 bpm. No P waves are seen before or after any QRS complex. However, there are fine and rapid undulations (↓) of the baseline characteristic of atrial fibrillation. The QRS complex duration is increased (0.18 sec), and there is a morphology that resembles a left bundle branch block (LBBB) with a tall R wave in lead I (←) and a QS pattern in leads V1–V6 (→). However, there is an initial Q wave in lead aVL (↑), so that this is not truly a LBBB pattern. In addition, the QRS morphology is very different from that seen in ECG 73A. This is not a junctional rhythm with a rate-related conduction abnormality, as the rate is slower than that seen in ECG 73A. Hence these are ventricular complexes. The RR intervals are regular (↔). However, as the underlying rhythm is atrial fibrillation, the RR intervals should be irregularly irregular. The regularity indicates that there is complete heart block present with an escape ventricular rhythm. The presence of a ventricular escape rhythm indicates that the location of the complete heart block is below the AV node within the His-Purkinje system. The presence of a junctional escape rhythm would place the block within the AV node. In this patient who had underlying bifascicular disease, the remaining fascicle, *ie*, left posterior fascicle, is now also unable to conduct impulses. The fact that the conduction abnormality is within the His-Purkinje system and not the AV node means that the complete heart block is not related to the two AV nodal blocking drugs, the β-blocker and diltiazem. Therefore, since the complete heart block is not AV nodal and thus not related to drugs, it is likely to be permanent and a pacemaker will be required. ■

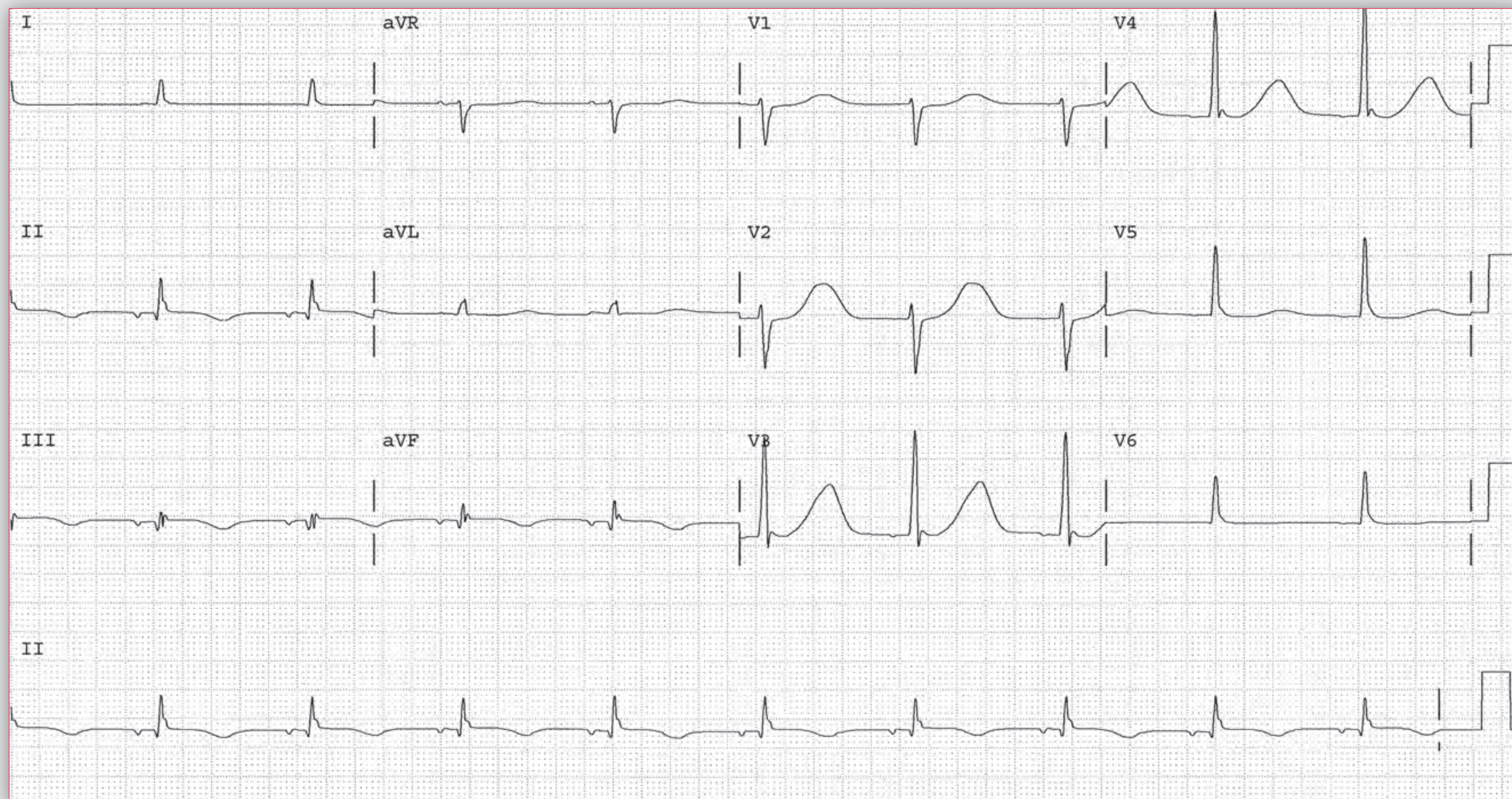
Notes

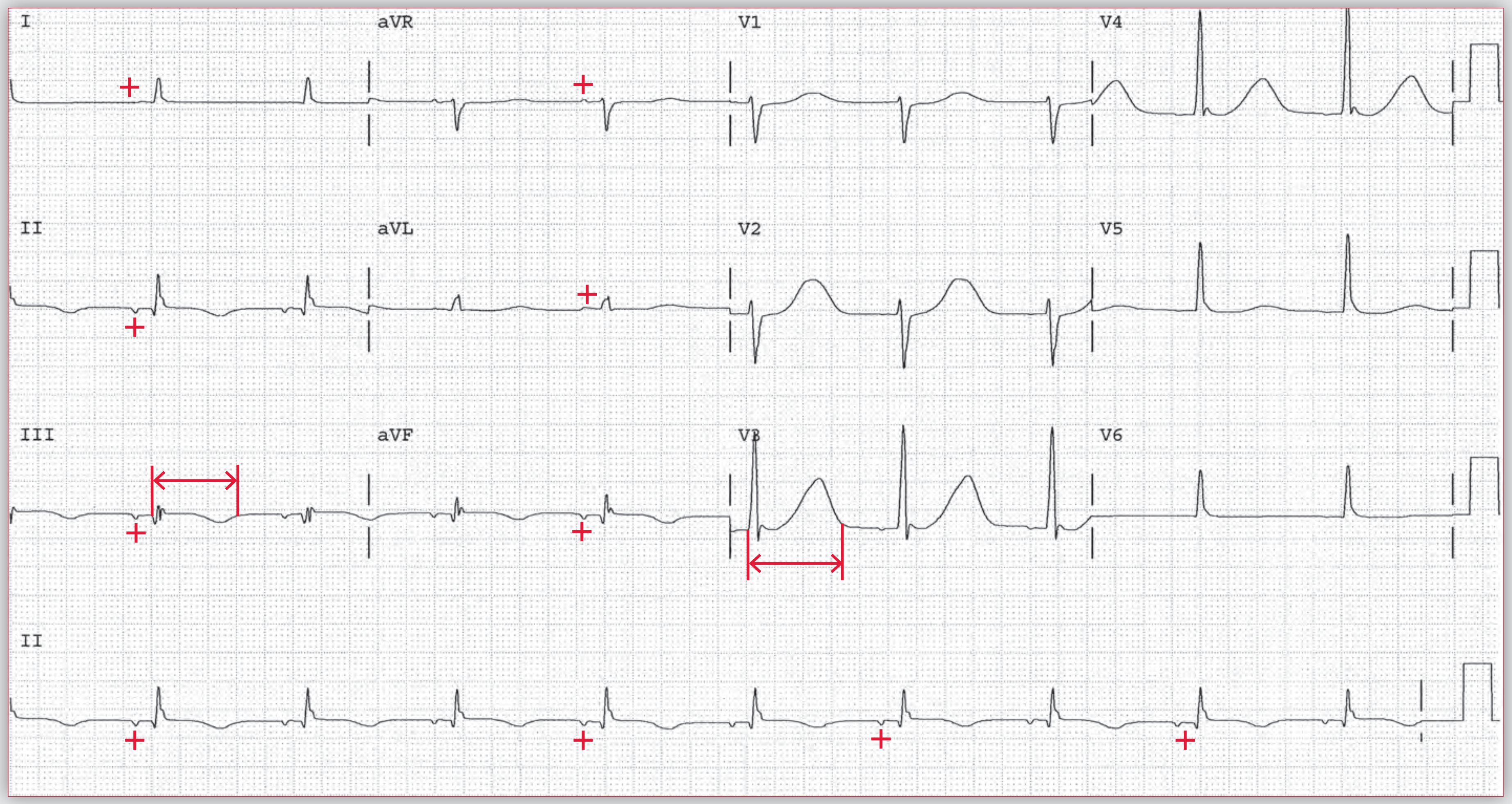
A 24-year-old HIV-positive patient treated with combination antiretroviral therapy presents to his primary doctor with 2 days of progressive headache and sinus pain. He is diagnosed with acute bacterial sinusitis and otitis media and prescribed erythromycin.

Several days later, he experiences unheralded syncope. On arrival at the hospital, an ECG is obtained.

What abnormality is seen?

What is the probably reason for syncope?





ECG 74 Analysis: Ectopic atrial rhythm, prolonged QT interval (long QT syndrome)

There is a regular rhythm at a rate of 58 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.14). However, the P wave is negative in leads II, aVF, and V3–V4. Therefore, this is not a sinus rhythm; it is an ectopic atrial rhythm.

The QRS complex duration is normal (0.10 sec) and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT interval is long (↔) (620 msec); the QTc is 610 msec. The prolonged QT interval is a result of a broad T wave and hence is called prolonged repolarization. This may be due to drugs, which is an acquired QT prolongation, or a genetic abnormality producing a channelopathy, which is a congenital long QT syndrome.

Based on the history and the use of a combination of drugs known to prolong the QT interval, the patient likely has an acquired long QT syndrome. The presence of a long QT interval is a risk factor for a polymorphic ventricular tachycardia that is termed torsade de pointes. This is a likely reason for the syncopal episode.

There is a long list of drugs that are associated with QT interval prolongation and hence a risk of torsades de pointes. Virtually all of the drugs that prolong the QT interval act by blocking a potassium current mediated by the potassium channel encoded by the HERG gene. This results in prolongation of phase 2 of the action potential. The proposed mechanism for drug-induced torsades de pointes is the development of early afterdepolarizations and triggered activity occurring during phase 2 of the action potential and resulting from prolonged repolarization. With a congenital prolonged QT interval, torsades is often induced by tachycardia. In contrast, torsades associated with the acquired form of a long QT syndrome is most commonly precipitated by bradycardia (bradycardic dependent) or by long-short RR intervals and hence called "pause-dependent" torsades.

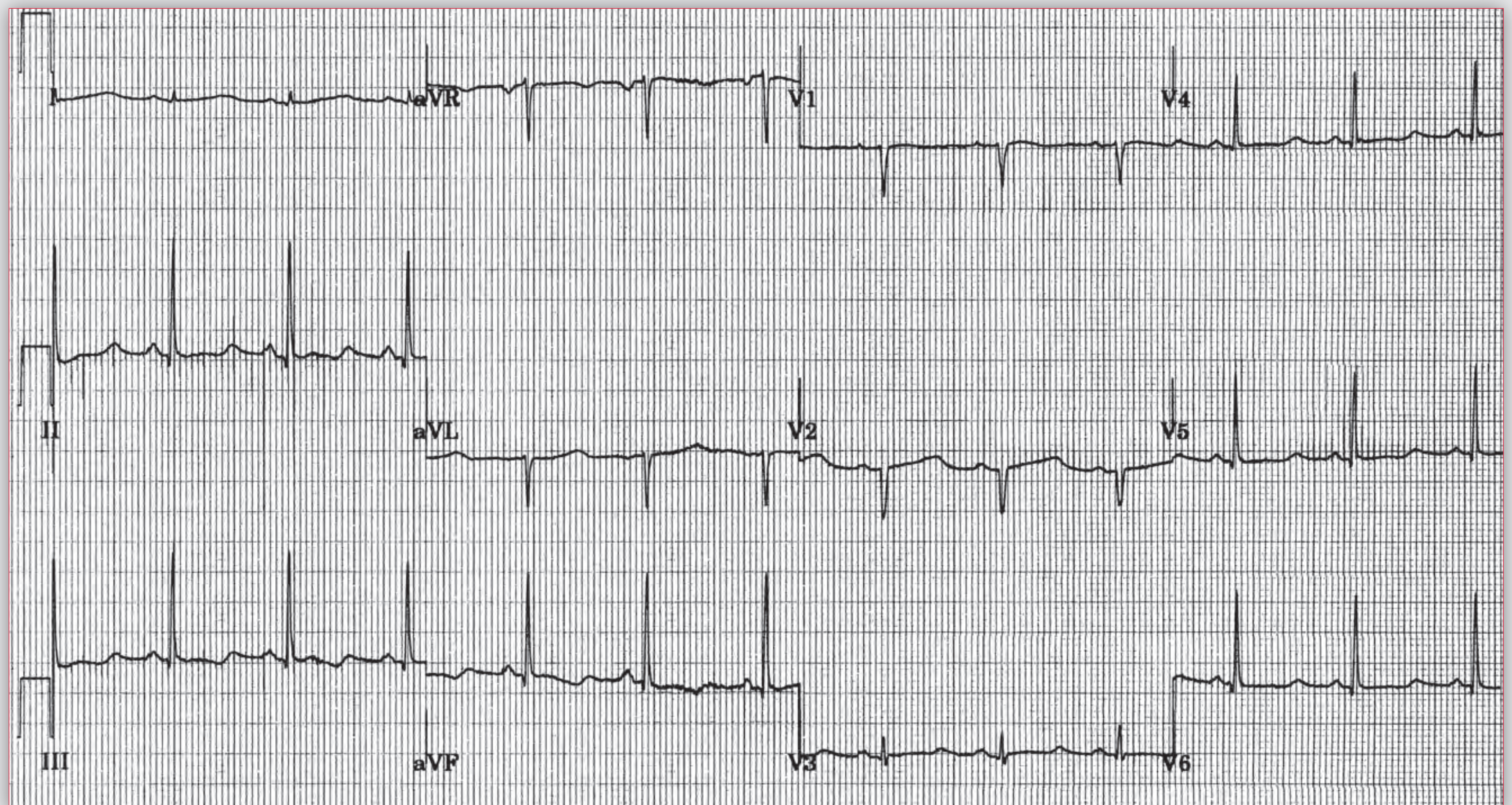
Treatment for torsades de pointes due to drug induced QT prolongation include:

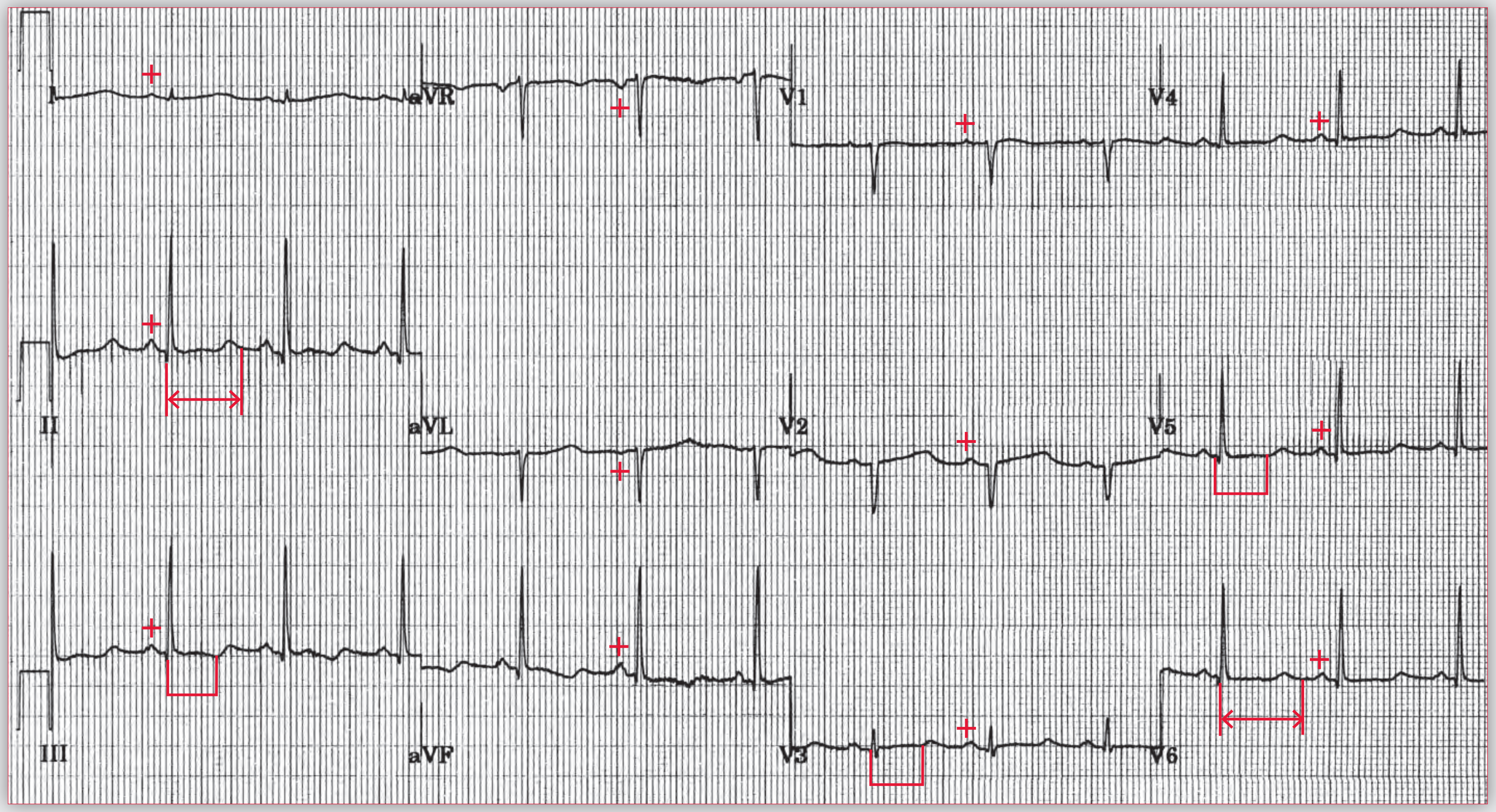
1. Discontinuation of drugs that are known to prolong the QT interval
2. Intravenous magnesium
3. Increasing the heart rate with the use of overdrive pacing; the increase in the heart rate will decrease the QT interval, due to a rate related shortening of His-Purkinje refractoriness.
4. Intravenous infusion of isoproterenol, which will increase the heart rate and shorten the QT interval, due to a rate related shortening of His-Purkinje refractoriness.
5. The use of class IB agents, such as lidocaine or mexiletine, may be of benefit. ■

Notes

A 62-year-old female undergoes thyroid resection for follicular cancer. On postoperative day 1, she complains of perioral numbness. During her bedside exam, she displays spasms of her feet and hands and loses consciousness. As part of her workup, and ECG is obtained.

What abnormality is noted on the ECG and what is the likely underlying etiology?





ECG 75 Analysis: Sinus rhythm, prolonged QT interval

There is a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a sinus rhythm.

The QRS duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in lead I and aVF). There is poor R-wave progression in V1–V2. This may be due to an old anterior septal myocardial infarction or could be a normal variant. The QT interval is prolonged (↔) (580 msec) with a rate corrected QT interval (QTc) of 640 msec. The QT prolongation is the result of a long ST segment (⊔), and hence this is delayed repolarization and not prolonged repolarization as the T wave has a normal duration. This form of long QT interval is seen with metabolic abnormalities, particularly low calcium or magnesium.

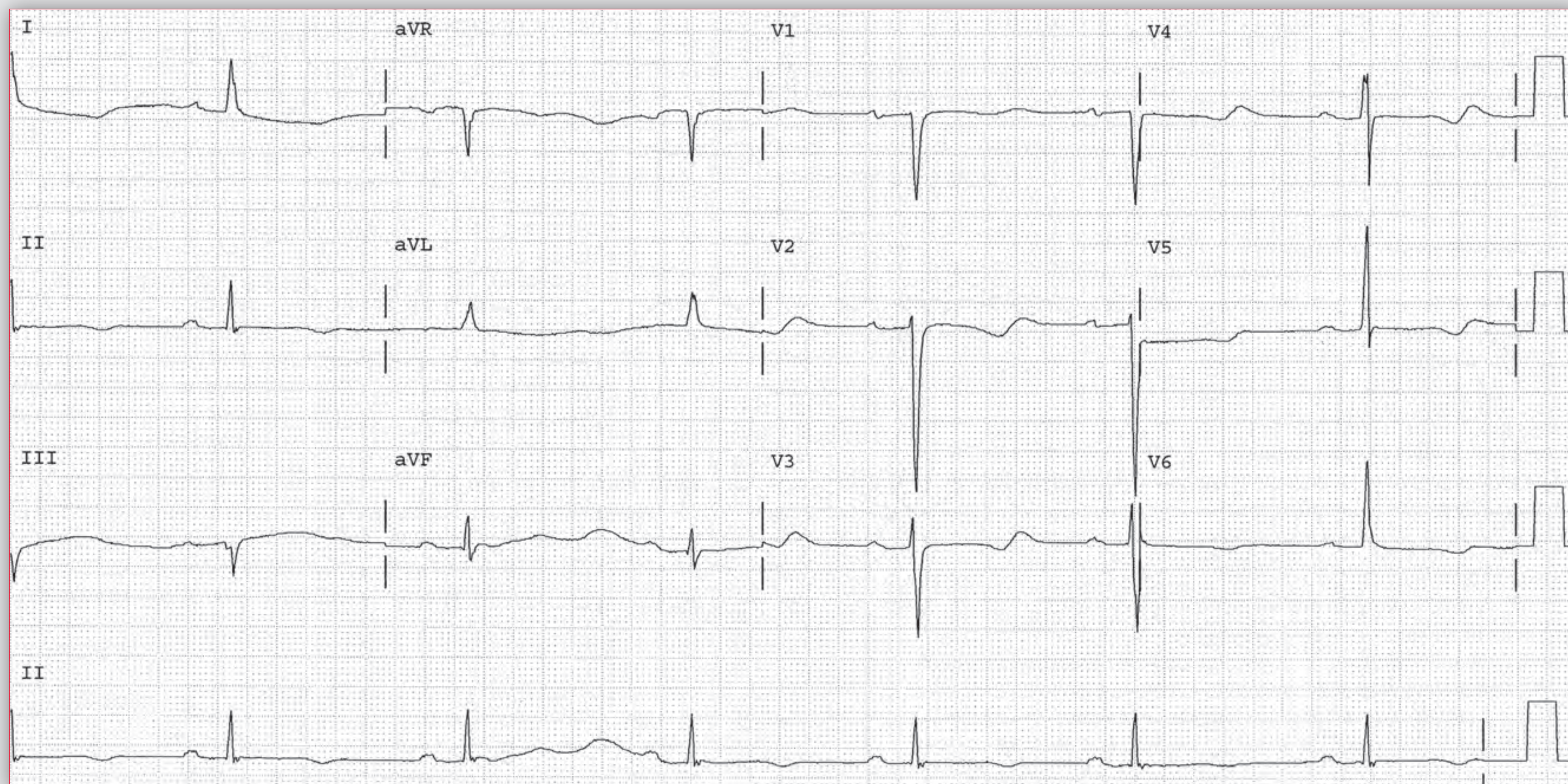
This patient has symptoms associated with hypocalcemia that is the result of hypoparathyroidism resulting from thyroid surgery. Although there is a prolonged QT interval, the occurrence of arrhythmia is very uncommon when the etiology is hypocalcemia. ■

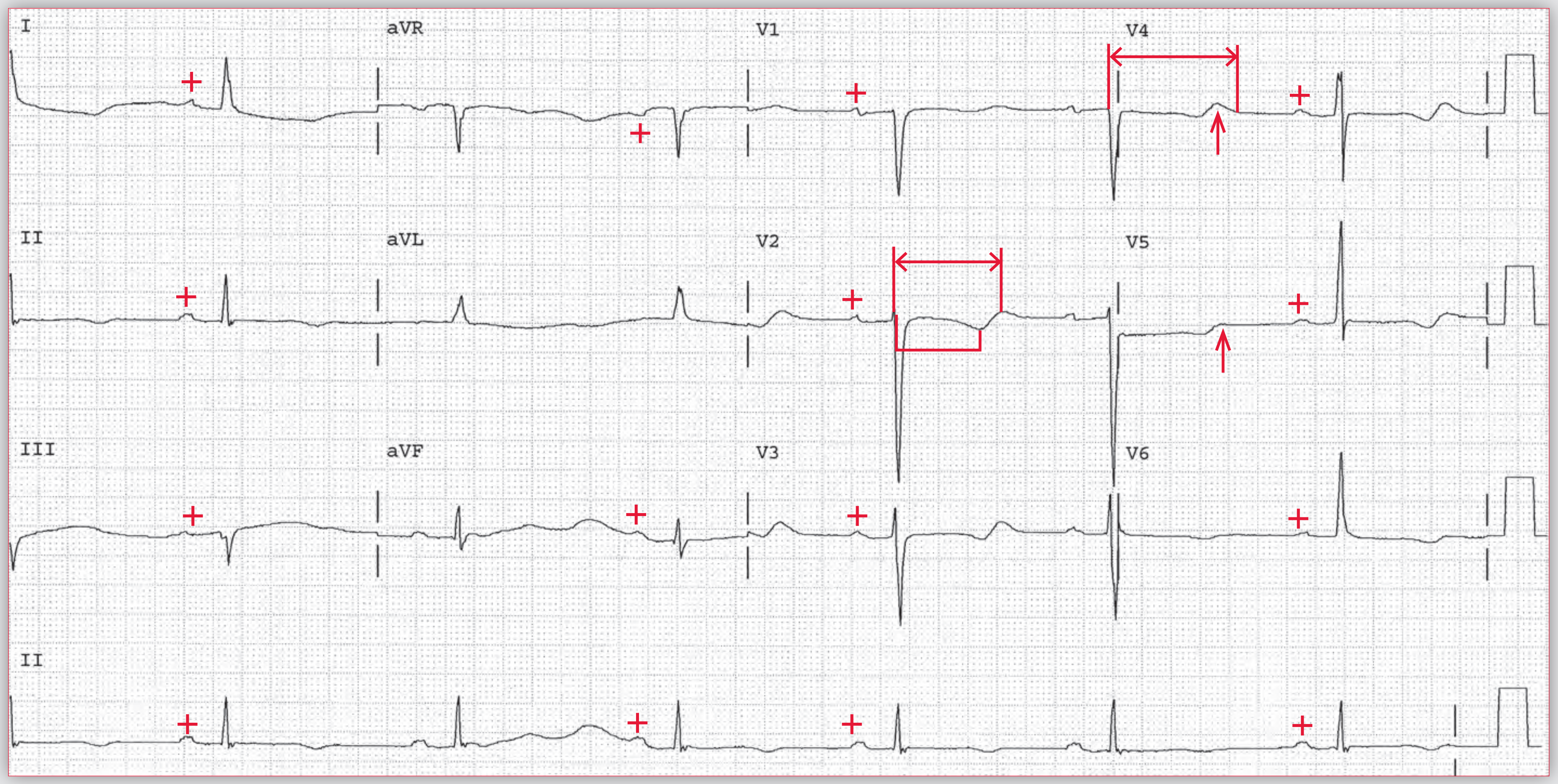
Notes

Core Case 76

A homeless male of unknown age is brought to the emergency department having been found unconscious by local law enforcement. He is disheveled and has poor hygiene. The odor of alcohol is apparent. He is nonresponsive but breathing spontaneously, albeit with a reduced respiratory rate. His vital signs are also notable for bradycardia. Laboratory values document a low serum magnesium but otherwise normal renal and liver function. Macrocytic anemia is noted. An ECG is obtained.

What abnormalities are noted, and what may be the cause of these abnormalities?





ECG 76 Analysis: Sinus rhythm, prolonged QT interval

There is a regular rhythm at a rate of 40 bpm. There is a P wave (+) before each QRS complex and the P wave is positive in leads I, II, aVF, V4–V6 and negative in lead aVR. Hence this is a sinus rhythm. The QRS complex duration is normal (0.10 sec) and the QRS complex morphology is normal. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF).

The QT interval is long (↔) (840 msec) and the QTc is 680 msec. The prolonged QT interval is the result of a long ST segment (⊔) while the T wave duration (↑) is normal. Therefore, this is delayed repolarization and not prolonged repolarization as the T wave has a normal duration. This form of long QT interval is seen with metabolic abnormalities, particularly low calcium or magnesium. Hypomagnesemia is common in alcoholic patients. Unlike a prolonged QT interval due to prolonged repolarization that is associated with torsades de pointes, the long QT interval due to hypomagnesemia is not associated with torsades de pointes. ■

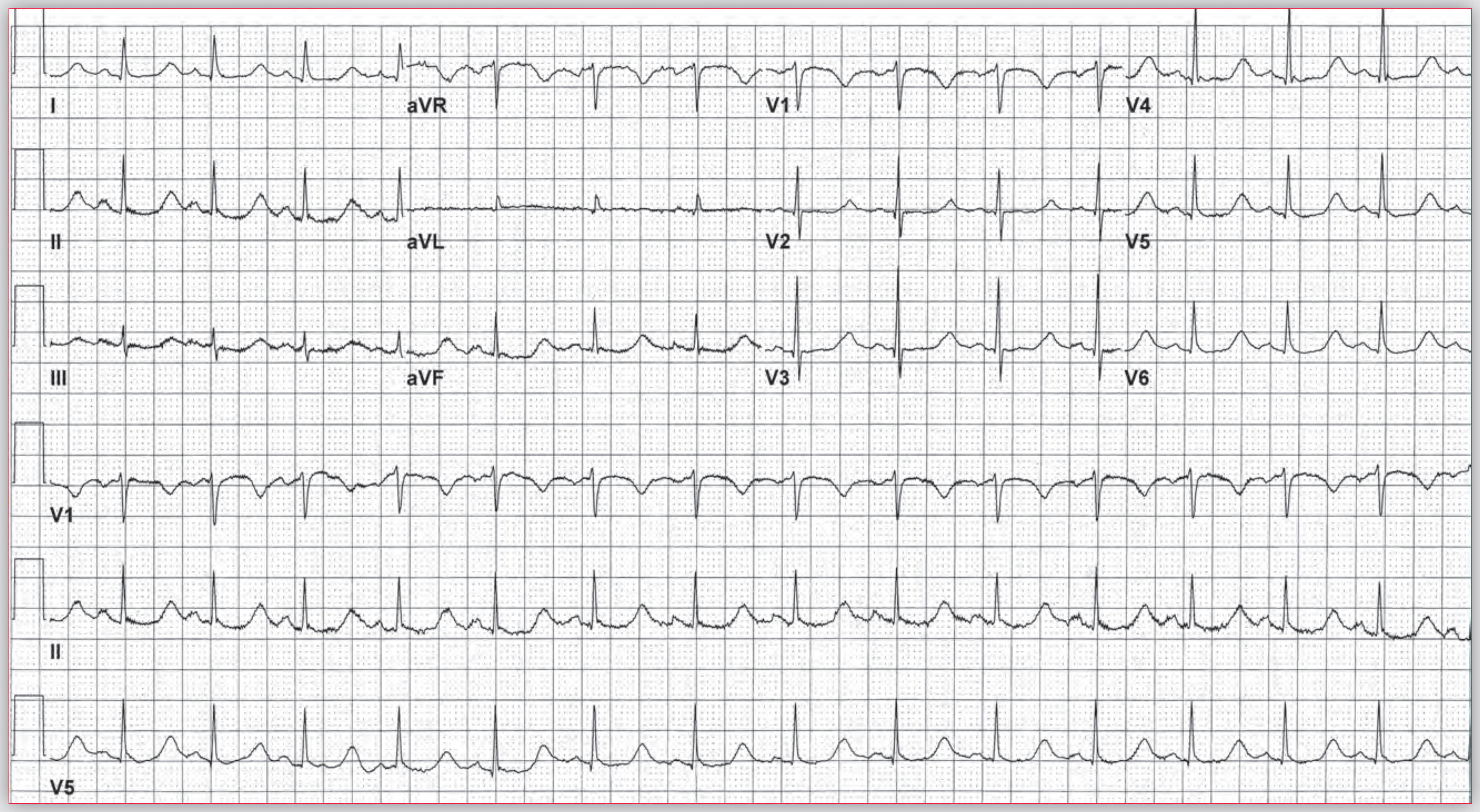
Core Case 77

A 44-year-old female is admitted to the intensive care with systemic inflammatory response syndrome and distributive shock secondary to acute pancreatitis.

A surveillance ECG is obtained (ECG 77A).

In the subsequent hours, she is treated appropriately and a repeat ECG is obtained (ECG 77B).

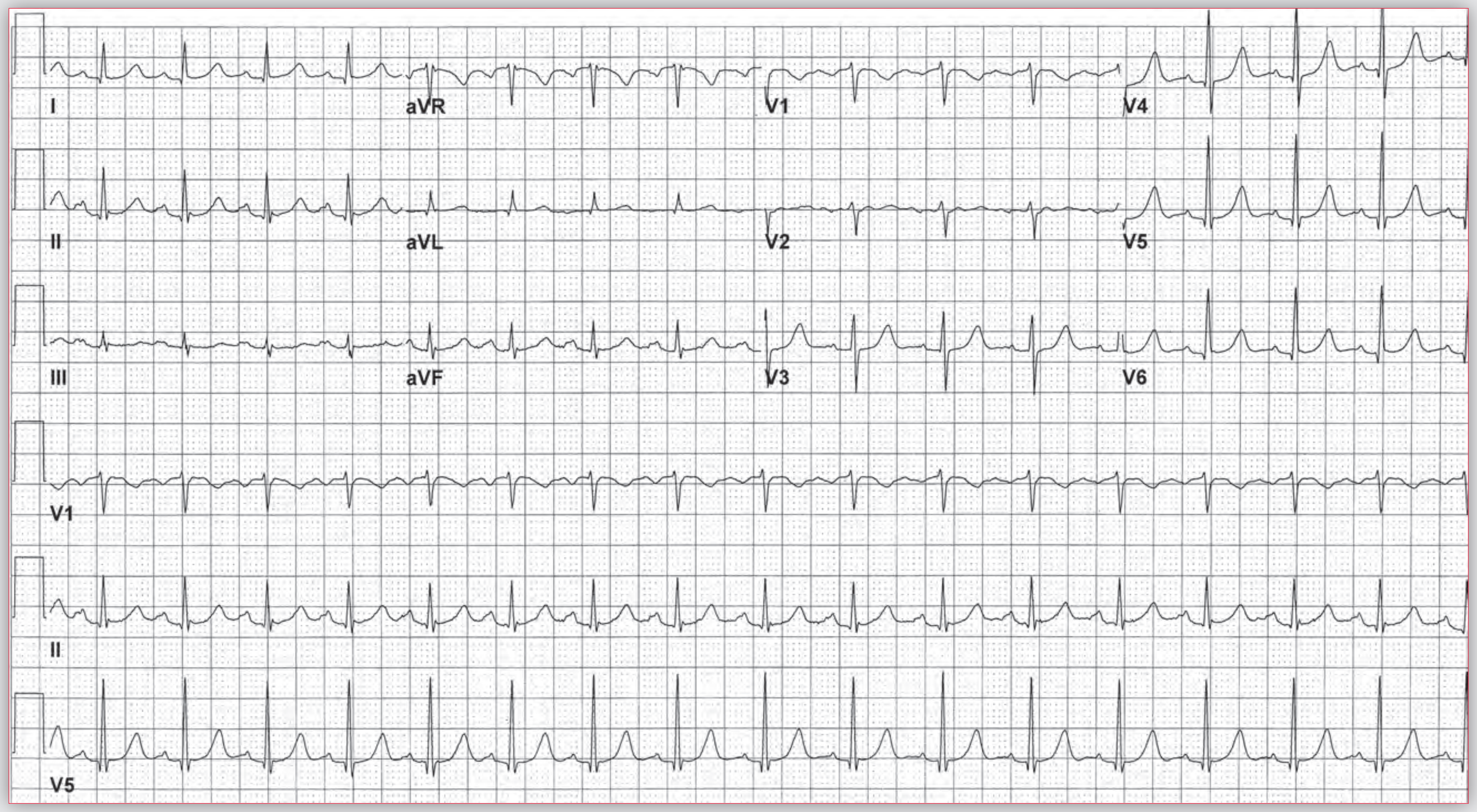
ECG 77A

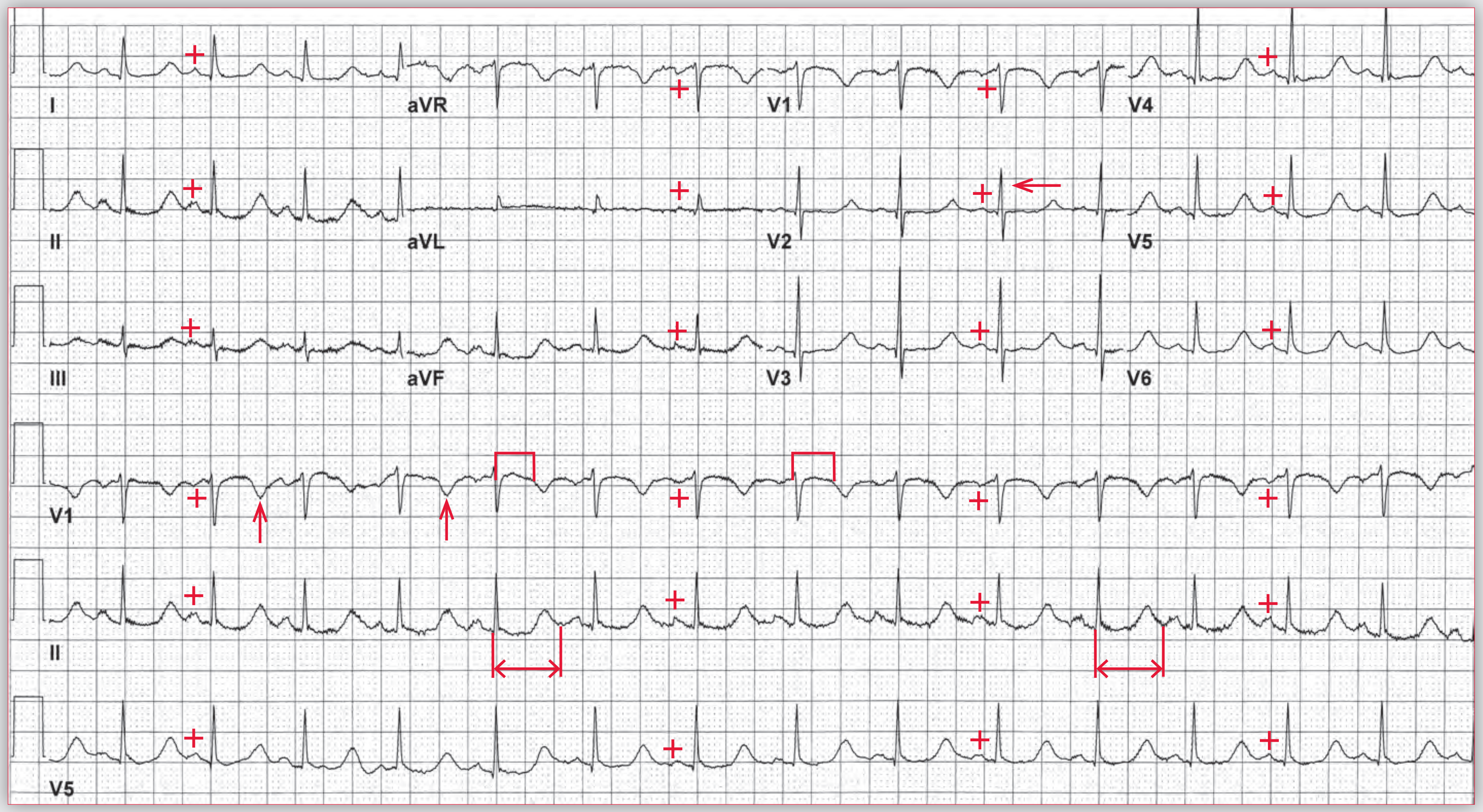


What abnormality is noted?

What is its relation to her clinical condition?

ECG 77B





ECG 77A Analysis: Sinus rhythm, early precordial R-wave transition (counterclockwise rotation), prolonged QT interval

ECG 77A shows there is a regular rhythm with a rate of 88 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. This is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and the axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). There is a normal morphology with early transition, *ie*, a tall R wave (←) in lead V2. This is due to counterclockwise electrical rotation in the horizontal plane, which is determined by imagining looking at the heart from under the diaphragm. With counterclockwise rotation, left ventricular forces are generated early and are seen in the anterior precordial leads, producing the tall R wave in lead V2.

The QT interval is prolonged (480 sec) (↔) and the corrected QT (QTc) is 580 msec. The prolonged QT is the result of a long ST segment (⊓) rather than a broad T wave; the T wave has a normal duration (↑). This is therefore delayed repolarization and not prolonged repolarization as the T wave has a normal duration. This form of long QT interval is seen with metabolic abnormalities, particularly low calcium or magnesium. In this case, the serum calcium was 5.5 and magnesium was 0.6.

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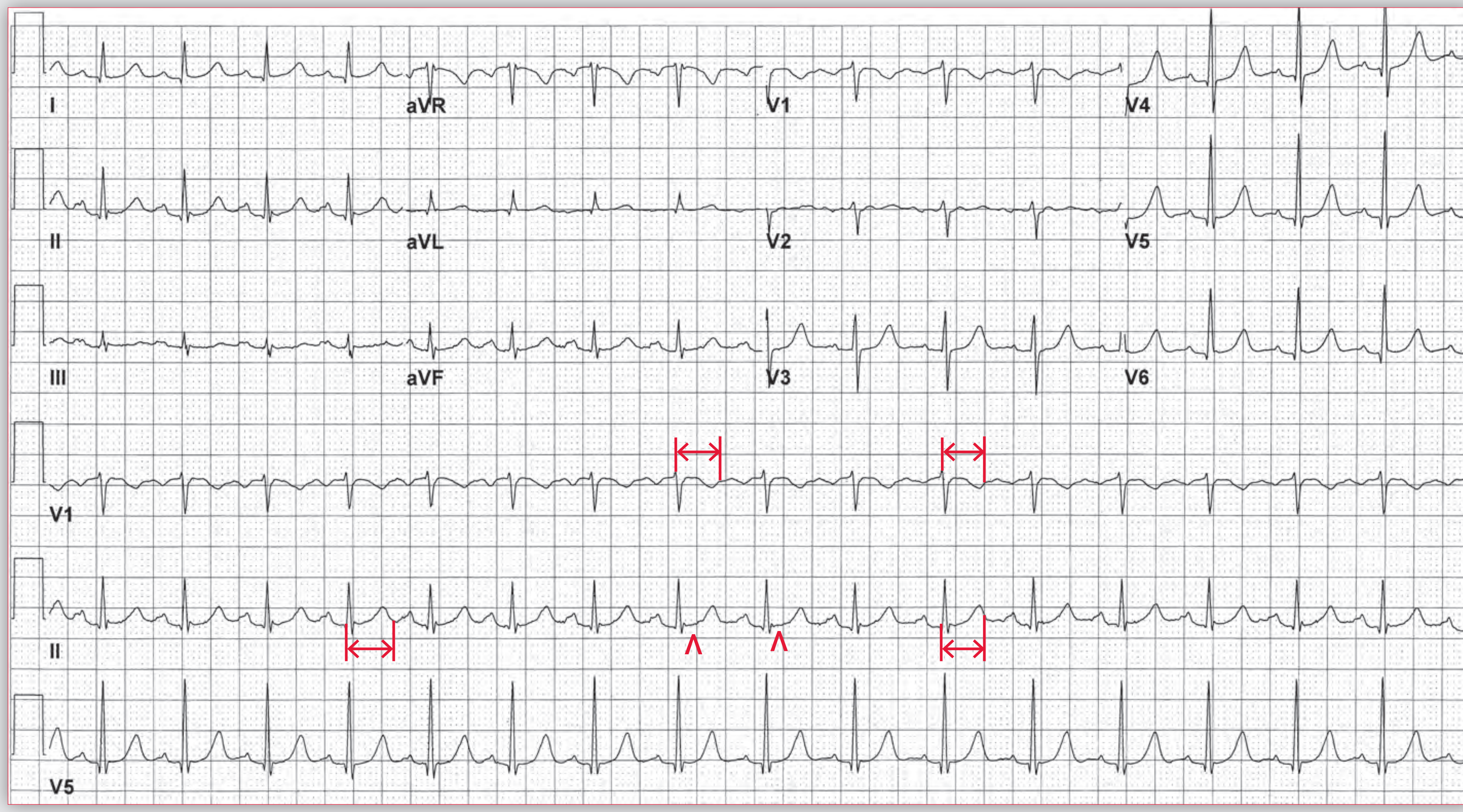


ECG 77B Analysis: Sinus tachycardia

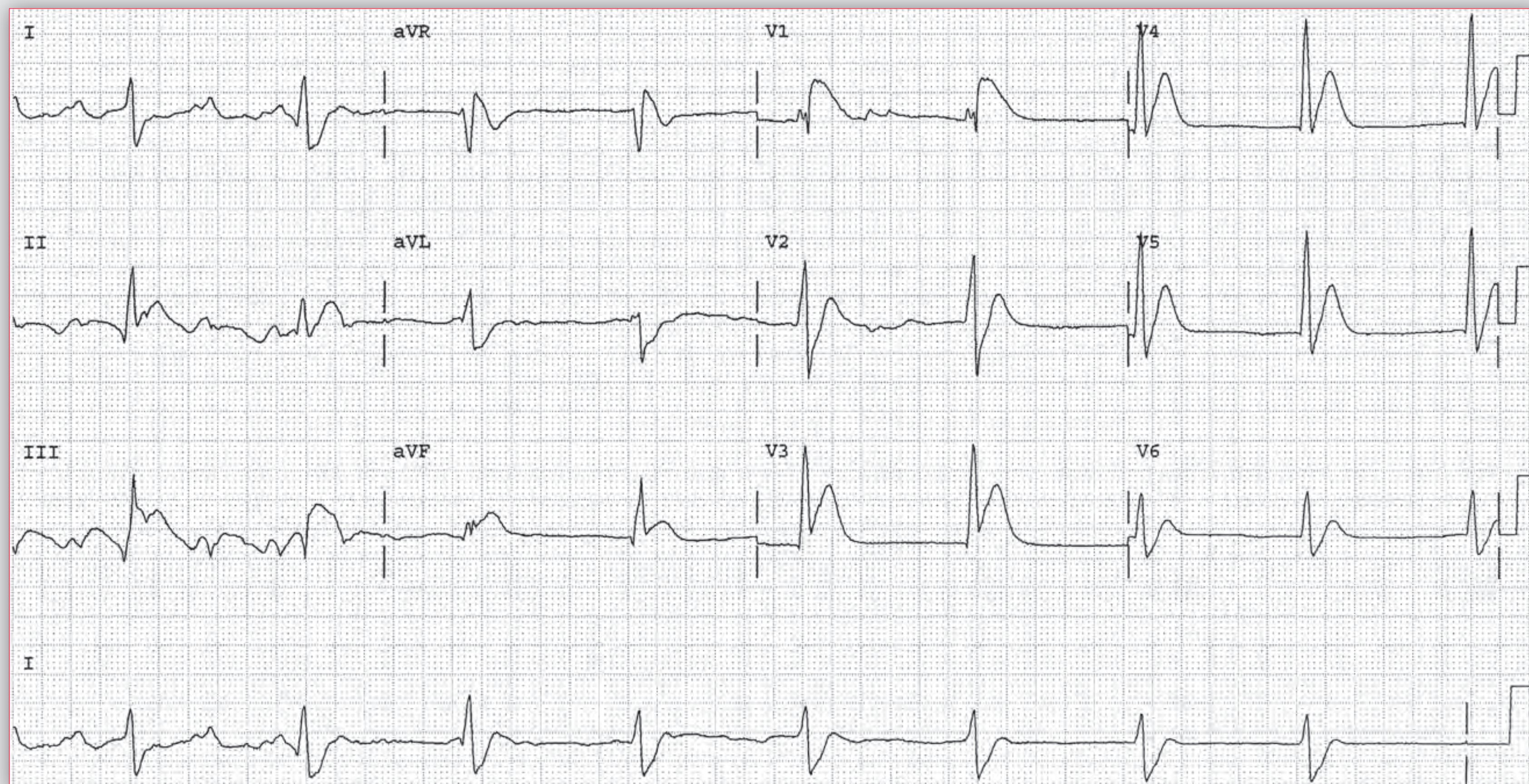
ECG 77B is from the same patient as ECG 77A and was recorded after correction of the serum calcium (8.9 mg/dL) and magnesium (1.9 mg/dL). The rhythm is regular with a rate of 100 bpm. The P wave, PR interval, QRS complex are identical to those of ECG 77A. Hence this is a sinus tachycardia. However, the ST segment (^) has shortened and the QT/QTc intervals (↔) are now normal (340/440 msec). ■

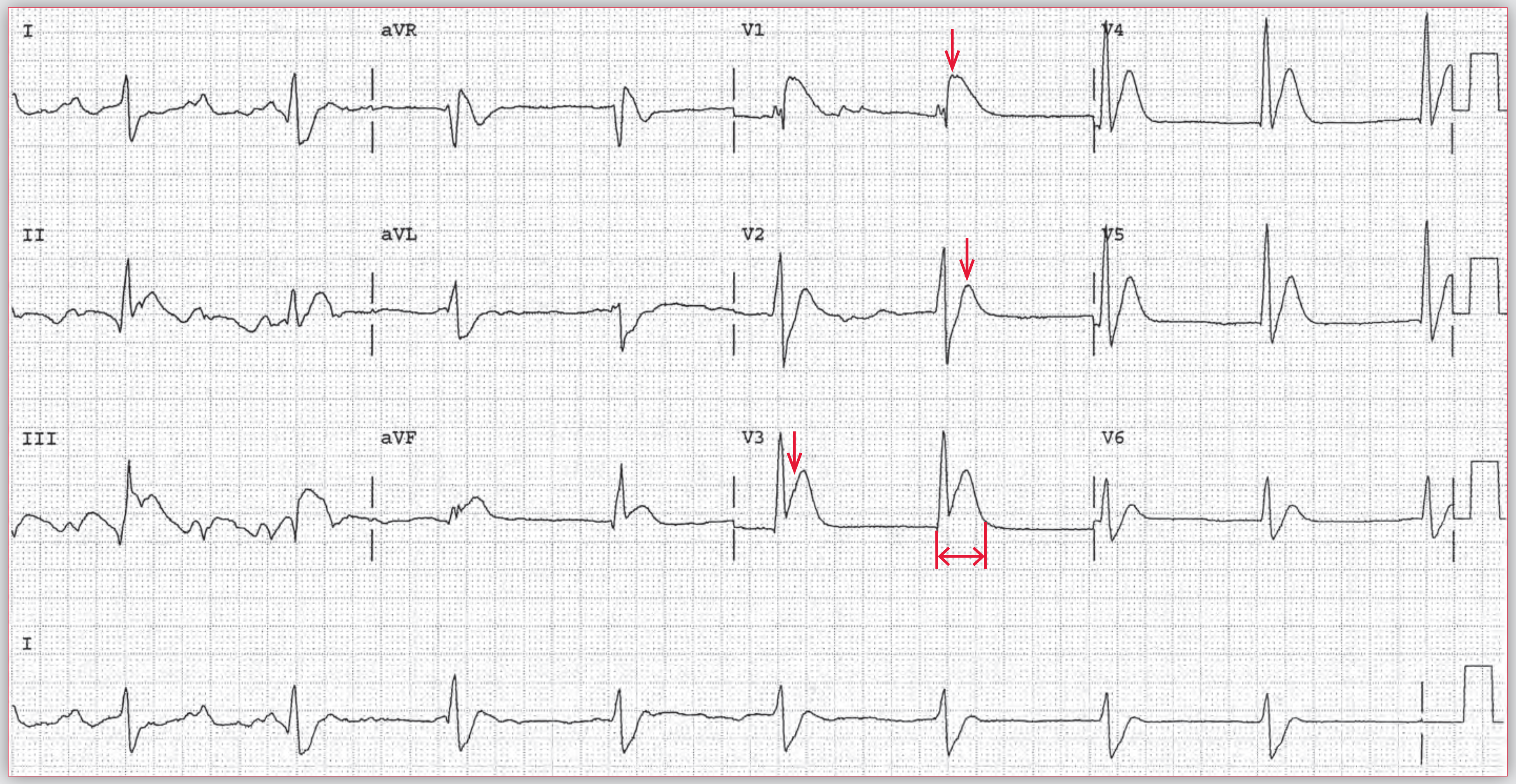
Notes

Core Case 78

A 64-year-old woman presents to her internist because of diffuse bone pain, fatigue, and a 20-pound weight loss. Routine blood tests showed a hematocrit of 25, a serum creatinine of 2 mg/dL, and an elevated calcium of 12 mg/dL. Subsequently, serum and urine protein electrophoresis (SPEP/UPEP) are ordered and the findings are consistent with a diagnosis of multiple myeloma. An ECG is obtained.

What abnormality is present?





ECG 78 Analysis: Junctional rhythm, early precordial R-wave transition (counterclockwise rotation), short QT interval

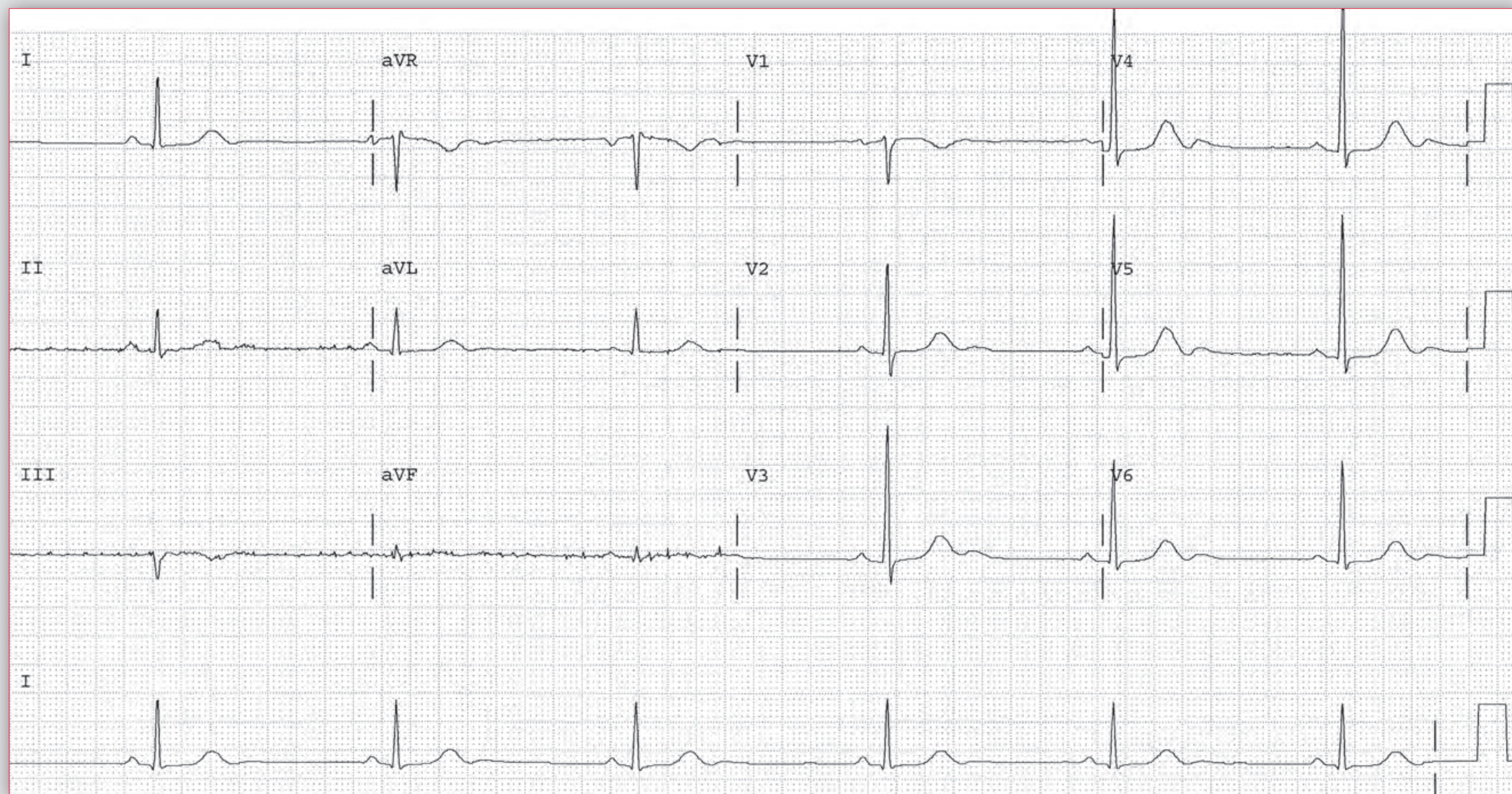
There is a regular rhythm with a rate of 52 bpm. There is no P wave seen before or after any QRS complex. The QRS complex duration is normal (0.10 sec), best measured in lead V4, and the morphology is normal. Therefore, this is a junctional rhythm. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT interval (↔) is 0.34 sec, and when corrected for heart rate, the QTc is 0.32 sec and hence very short. This is due to the absence of any ST segment. In addition, there is the appearance of ST-segment elevation and an acute myocardial infarction, particularly in leads V1–V3 (↓).

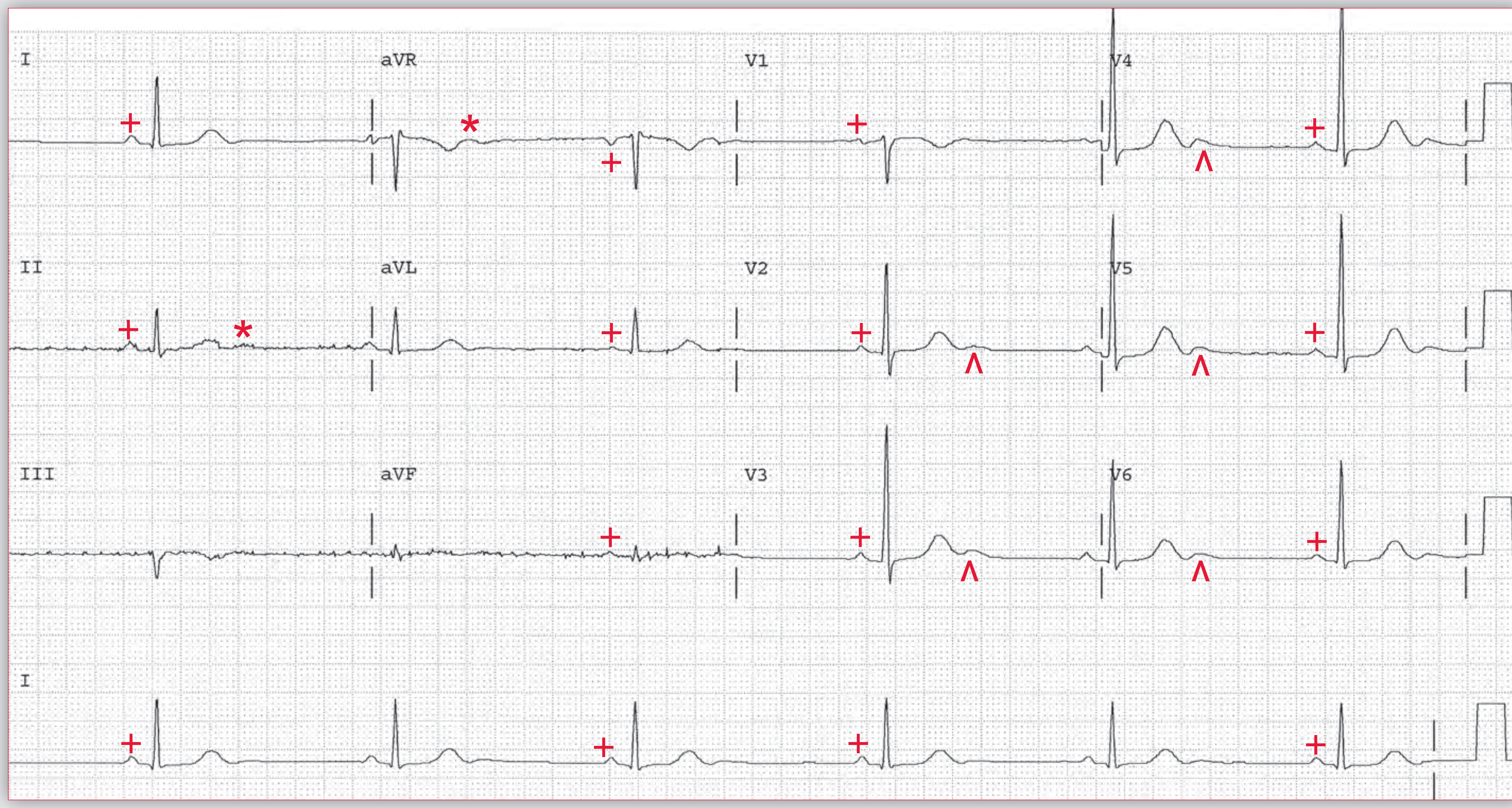
A short QT interval (defined as a QTc ≤ 0.34 sec) is due to metabolic abnormality (high calcium or magnesium) or a congenital short QT syndrome that is due to a channelopathy involving the sodium, potassium, or calcium channels. The congenital short QT syndrome is associated with an increased risk of sudden death due to ventricular fibrillation. In this case, the short QT interval is the result of hypercalcemia, which is due to multiple myeloma. Usually there are no arrhythmic problems associated with hypercalcemia. ST-segment elevation mimicking myocardial infarction has been reported with hypercalcemia; it is seen in this patient. It is likely that the apparent ST segment elevation is actually the T wave that is at the very end of the QRS complex as a result of the short ST segment. ■

Notes

A 26-year-old woman is admitted to the hospital with viral gastroenteritis and marked volume depletion secondary to vomiting. An ECG obtained the emergency department is abnormal.

What is the underlying abnormality?





ECG 79 Analysis: Sinus bradycardia, prominent U waves

There is a regular rhythm with a rate of 36 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, V4–V6. Hence this is a sinus bradycardia.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (QRS positive in leads I and aVF). Following the T wave, there is another positive waveform seen in lead V2–V6. These waveforms can also be seen in leads II and aVL (*). These are prominent U waves (^), which are suggestive of hypokalemia. The QT/QTc intervals are normal (550/430 msec). As the U wave follows the T wave, it is not included in the measurement of the QT interval.

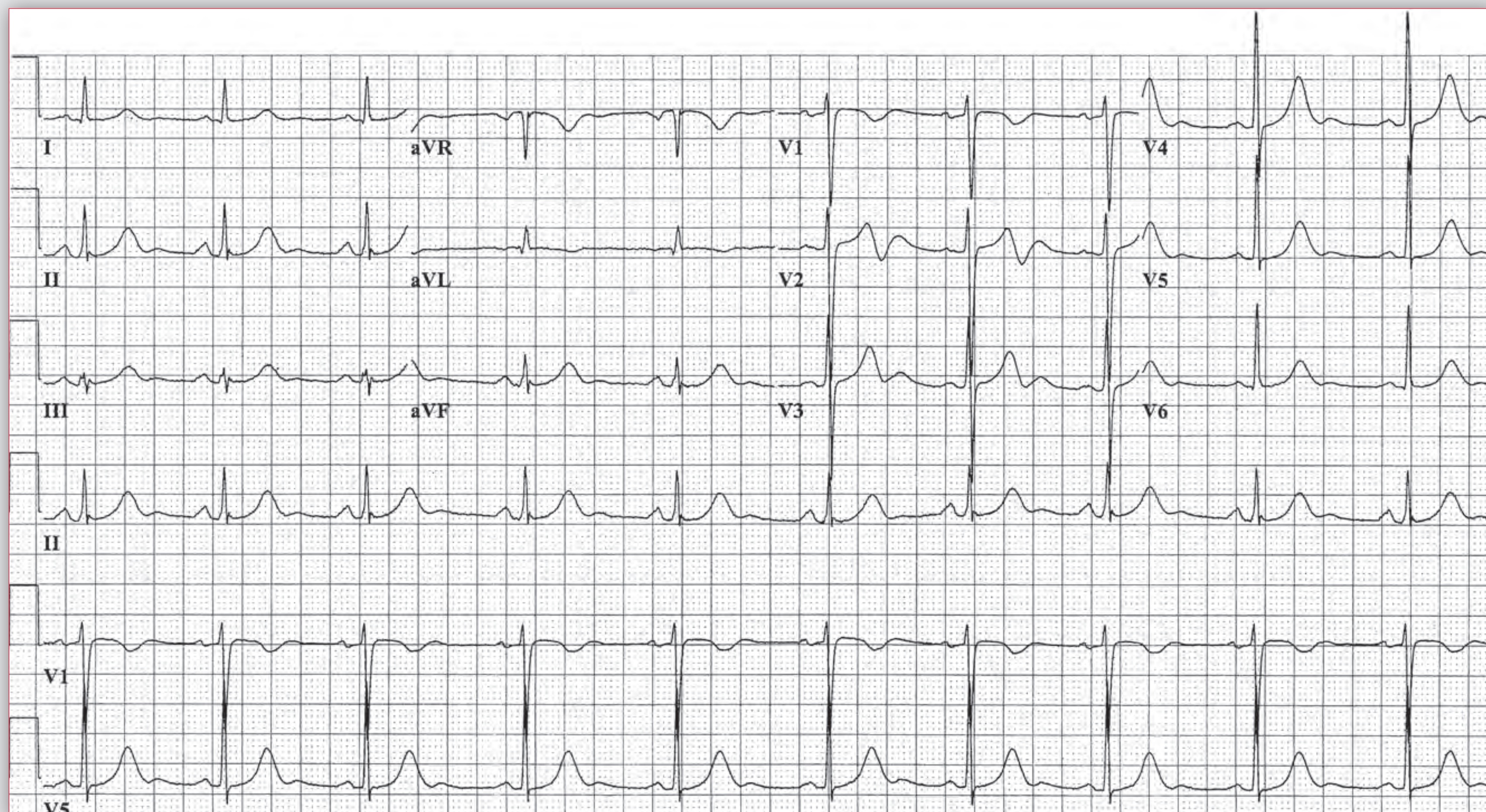
The U wave is normally seen in right precordial leads and is felt to represent late or secondary repolarization of the His-Purkinje system or possibly the papillary muscles. In hypokalemia, the amplitude of the U wave is increased and is seen in most or all of the precordial leads. It may also be seen in the limb leads. However, prominent U waves may also be seen with bradycardia. ■

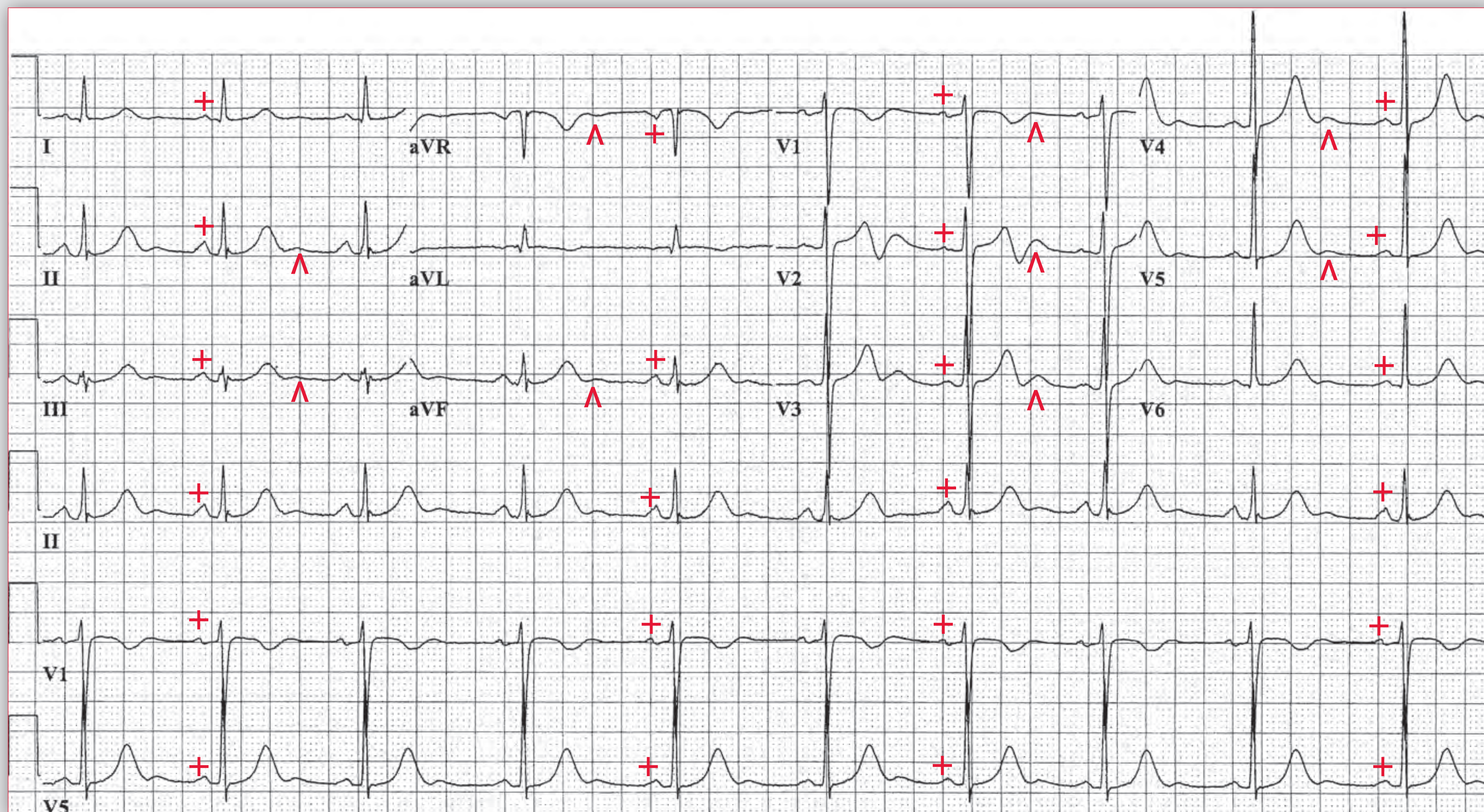
Notes

Core Case 80

A 75-year-old man with a history of peripheral edema secondary to venous insufficiency is begun on daily therapy with furosemide. One week later, he returns for a routine follow-up visit. Peripheral edema has decreased but is still present. As a result, his physician adds hydrochlorothiazide. Three days later, he is seen in the emergency department for complaints of fatigue and muscle cramps. Routine laboratory tests are obtained, and serum potassium is 2.8 mEq/L. An ECG is obtained.

Is the ECG normal?





ECG 80 Analysis: Normal sinus rhythm, prominent U waves

There is a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (QRS positive in leads I and aVF).

The QT/QTc is normal (440/440 msec). Following the T wave in leads V2–V6 as well as leads II, III, aVR, and aVF, there is a prominent positive waveform that is a U wave (^), which is indicative of possible hypokalemia.

The U wave is normally seen in right precordial leads and is felt to represent late or secondary repolarization of the His-Purkinje system or possibly the papillary muscles. In hypokalemia, the amplitude of the U wave is increased and is seen in most or all of the precordial leads. When the U wave follows the T wave, it is not considered part of the QT interval. ■

Notes

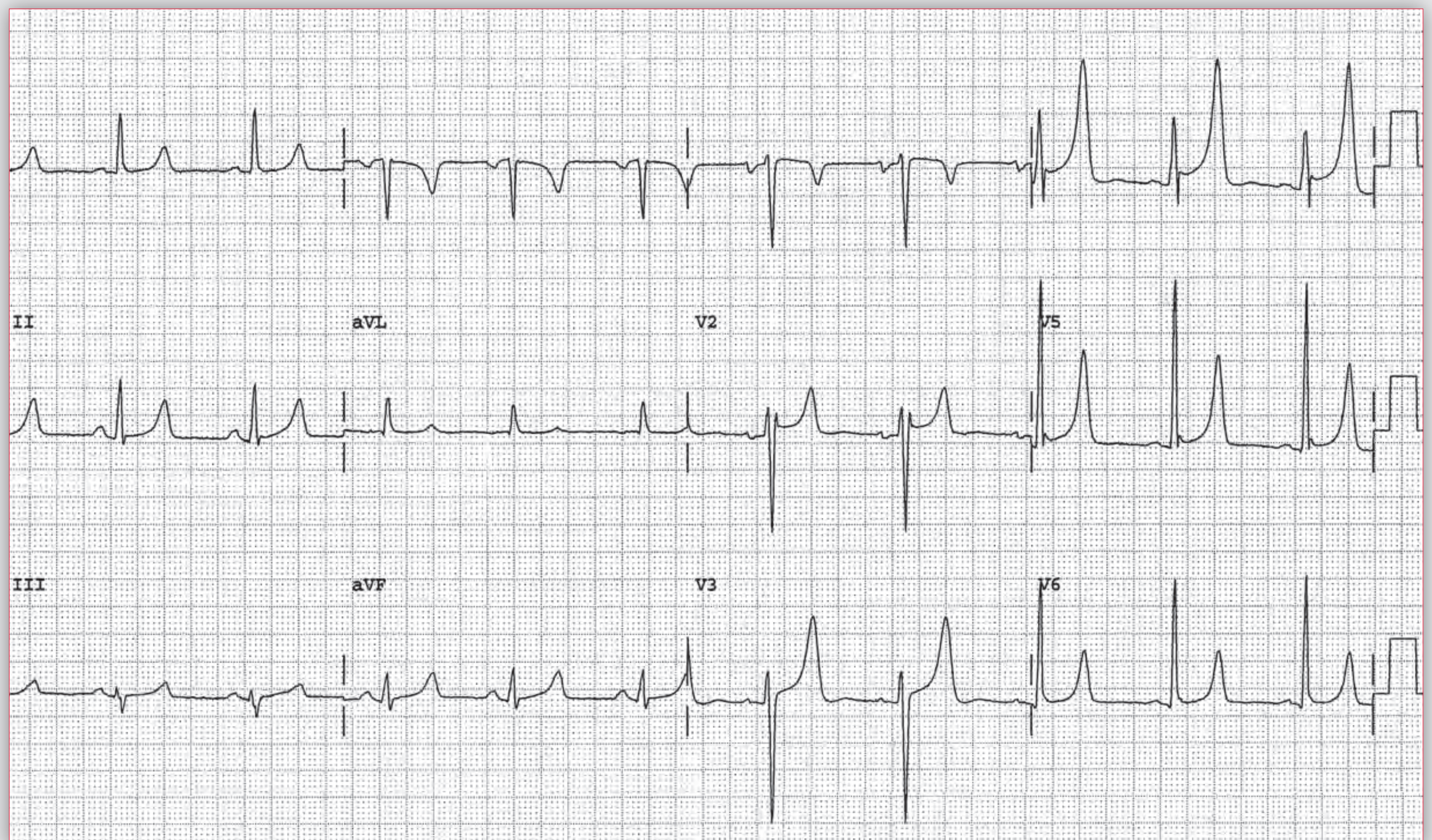
Core Case 81

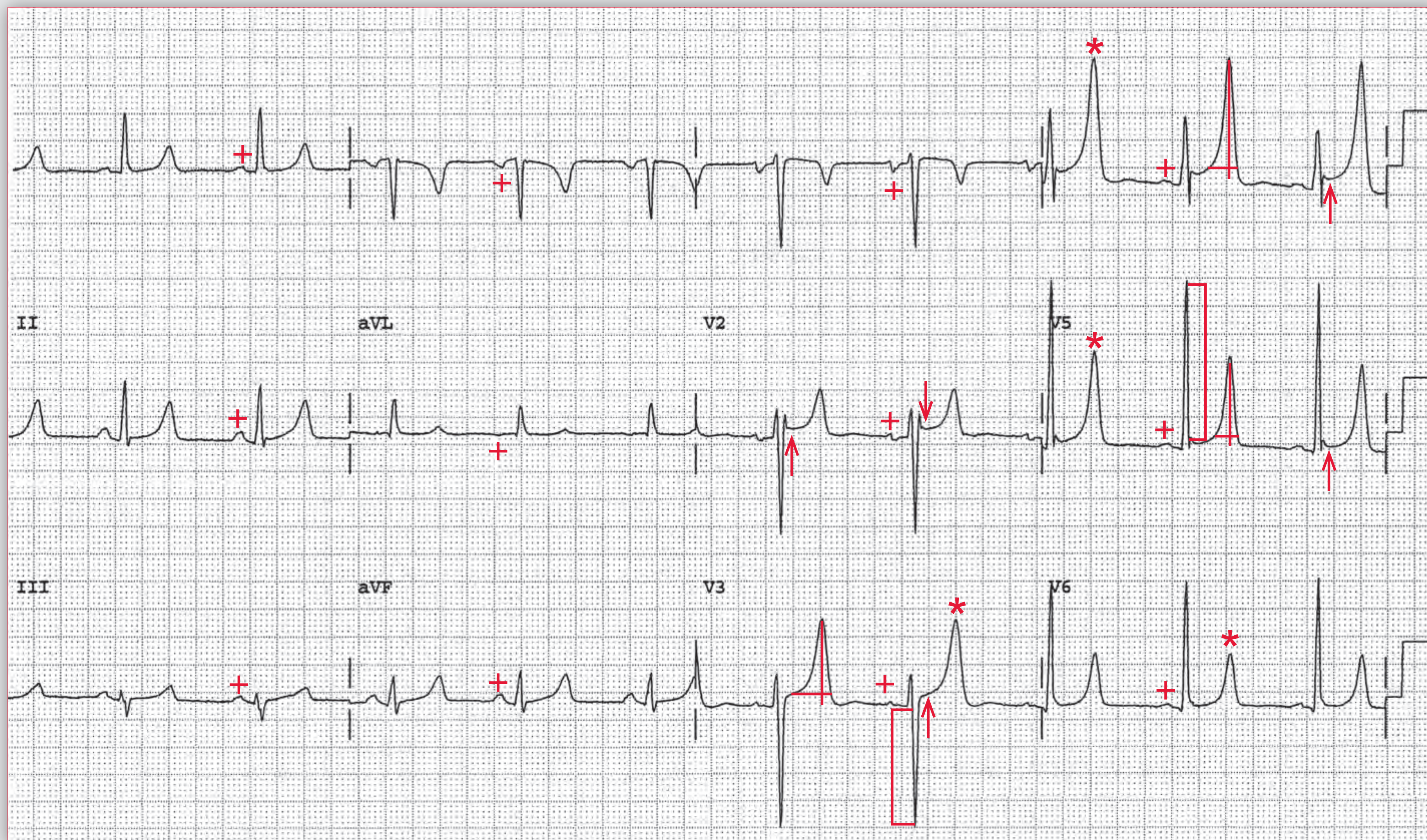
A 59-year-old man with bicuspid aortic valve and moderate to severe aortic stenosis is admitted with chest discomfort occurring with exercise. He has noted progressive decrease in his exercise tolerance over the past 3 months and has not had any pain at rest. His admission ECG is shown.

His providers are concerned about acute ischemia.

What are the key abnormalities?

Are the concerns of acute ischemia warranted?





ECG 81 Analysis: Normal sinus rhythm, left atrial hypertrophy, right ventricular conduction delay, left ventricular hypertrophy, early repolarization

There is a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex and the PR interval is stable (0.016 sec). The P wave is upright in leads I, II, aVF, and V4–V6. Therefore, this is a normal sinus rhythm. The P wave is negative in lead V1 and is wide and notched in lead II, consistent with left atrial hypertrophy.

The QRS complex duration is normal (0.10 sec). There is an RSR′ in lead V2 (↓), suggesting a right ventricular conduction delay. The QRS complex amplitude is increased in lead V5 ( ]) (30 mm or boxes) and the S wave depth in lead V3 ([ ) is increased (22 mm or boxes). Hence there are criteria for left ventricular hypertrophy (*ie*, S V3 + R V5 ≥ 35 mm). Associated with this is J point and ST elevation in leads V2–V4 (↑), which is termed early repolarization. The axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (460/460 msec).

The T waves are tall and peaked (*), particularly in leads V3–V6. However, the T waves are asymmetric with a slower upstroke and more rapid downstroke. Hence, these are normal T waves and likely are tall and peaked as a result of left ventricular hypertrophy.

The normal T wave is asymmetric, regardless of amplitude. The upstroke of the T wave is slower, while the downstroke of the T wave is faster. T waves that are tall, peaked, and symmetric with an upstroke and downstroke that are equal (hyperacute) are seen with hyperkalemia, which may be systemic or localized as in acute MI. In this case, the tall peaked T-wave abnormality as well as the ST-segment elevation could be confused with any early myocardial infarction. As indicated, the ST-segment elevation is early repolarization, which is often seen with left ventricular hypertrophy or increased QRS voltage. The patient's chest pain, which occurs only with exertion, is likely due to progressive aortic stenosis rather than an acute coronary syndrome. ■

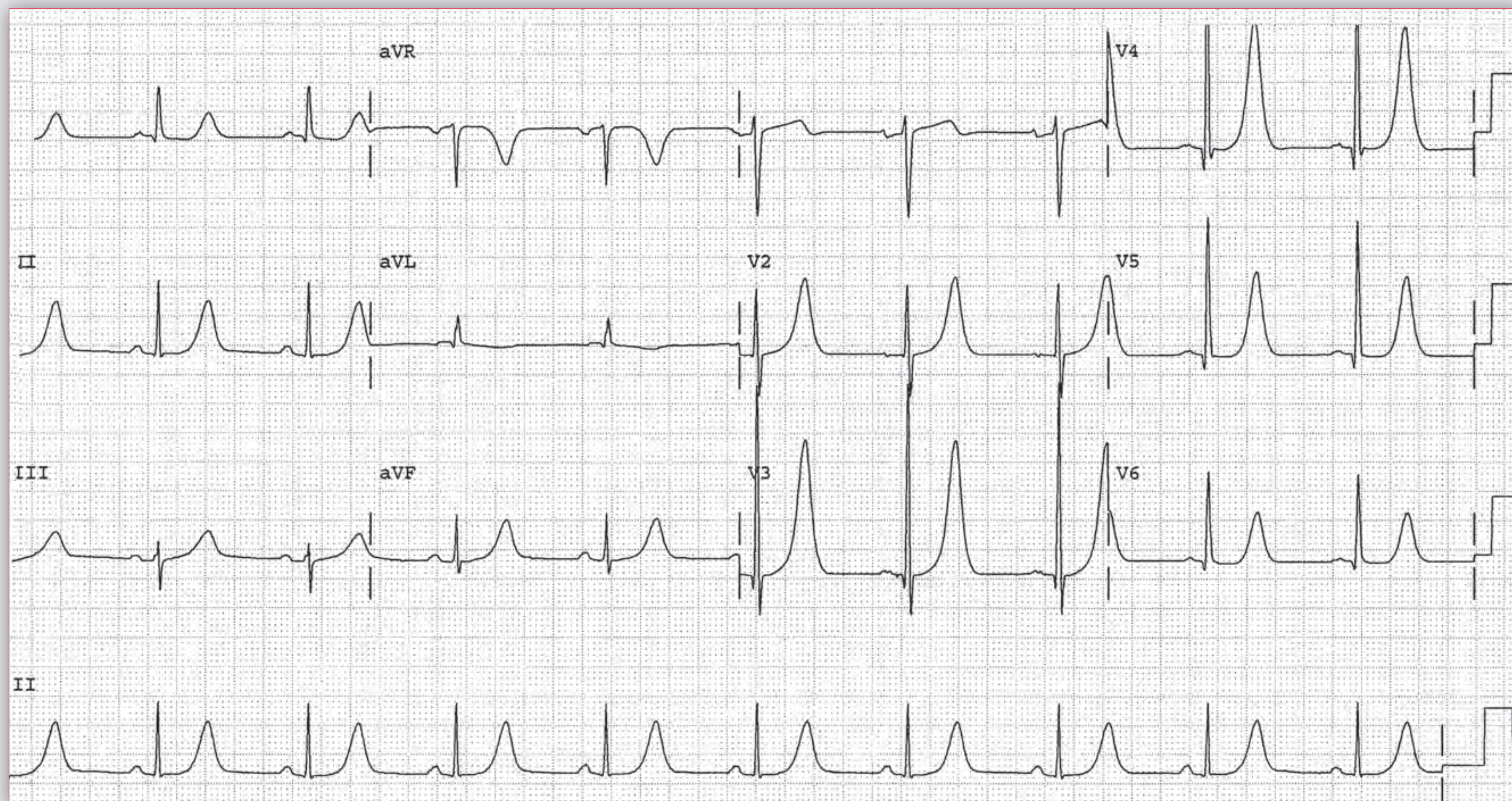
Notes

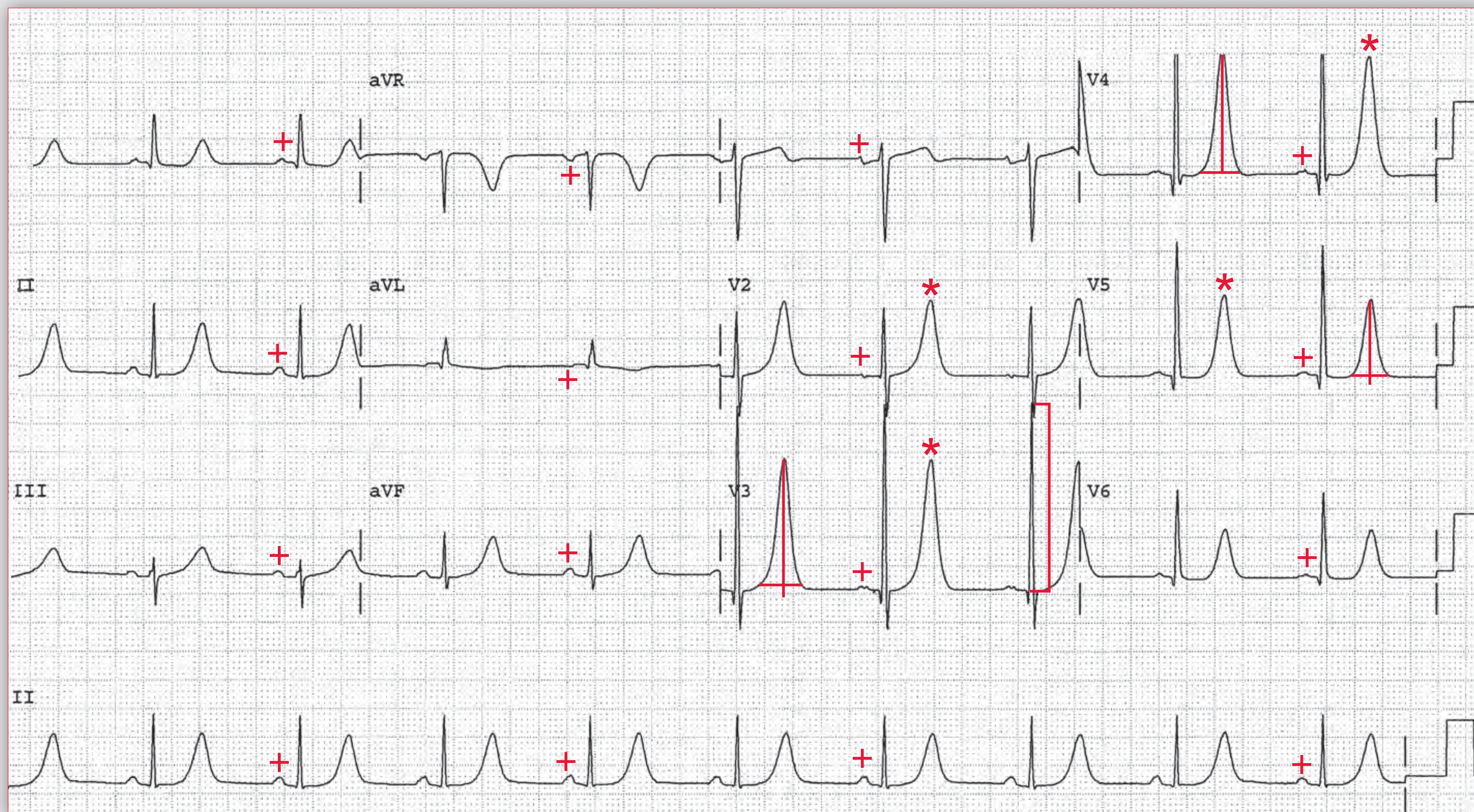
Core Case 82

A 68-year-old man with end stage renal disease is seen at his hemodialysis center. He missed his last scheduled hemodialysis session.

What does his ECG show?

What else can cause this pattern?





ECG 82 Analysis: Sinus bradycardia, left ventricular hypertrophy, hyperacute T waves

There is a regular rhythm at a rate of 56 bpm. There is P wave (+) before each QRS complex and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. The QRS complex duration is normal (0.08 sec). The R wave amplitude in lead V3 is 33 mm or boxes ( ]), meeting one of the criteria for left ventricular hypertrophy (*ie*, ≥ 25 mm in any precordial lead). The axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (480/460 msec).

The T waves are tall, peak and symmetric (*), especially prominent in leads V3–V5. The T wave morphology is called hyperacute and is seen with systemic hyperkalemia or localized hyperkalemia seen as an early change of an acute ST-segment elevation myocardial infarction.

The normal T wave is asymmetric, regardless of amplitude. The upstroke of the T wave is slower, while the downstroke of the T wave is faster. T waves that are tall, peaked, and symmetric (upstroke and downstroke equal) are seen with hyperkalemia, which may be systemic or localized as with an acute MI.

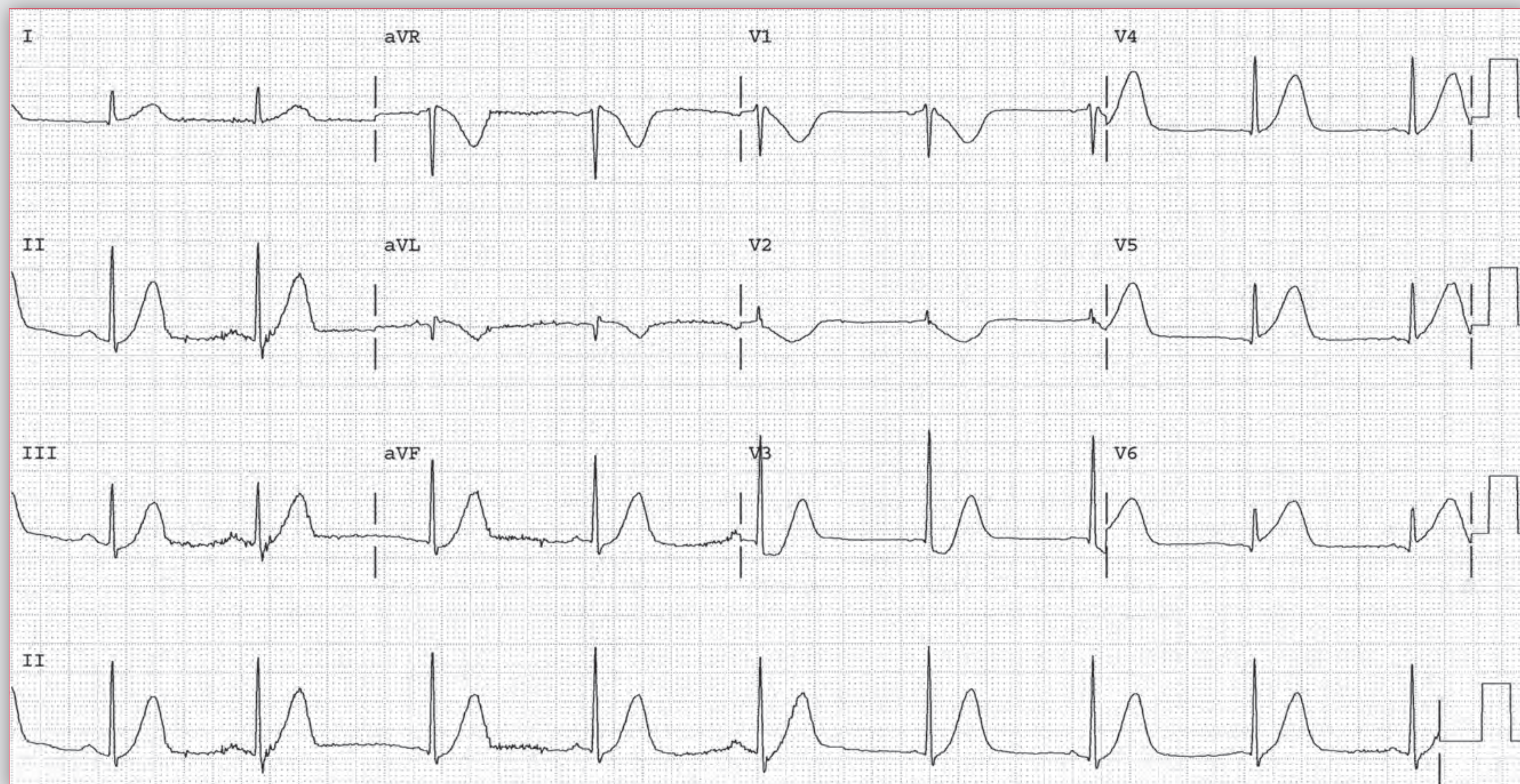
Notes

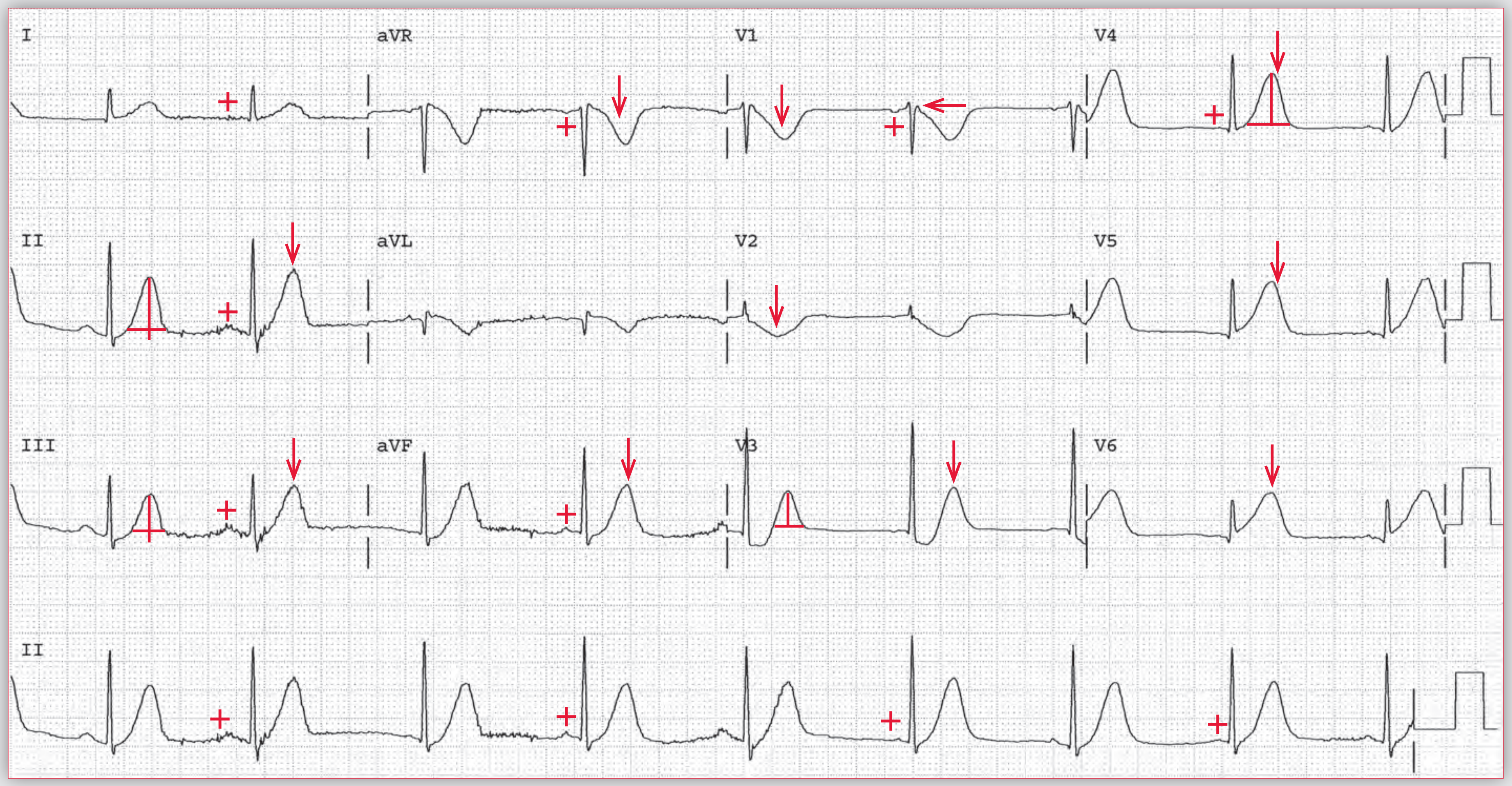
Core Case 83

A 62-year-old woman with type I diabetes, renal insufficiency, and hypertension has recently begun therapy with an ACE inhibitor. She is admitted with mental status changes. Her companion thinks she may have missed a few doses of insulin. Upon admission her serum glucose is 450 mg/dL, BUN is 76 mg/dL, and creatinine is 3 mg/dL, which are increased compared to her baseline. It is felt that as a result of hyperglycemia and the use of an ACE inhibitor she is severely dehydrated, acidotic, and has electrolyte abnormalities.

What does the ECG show?

What is the likely diagnosis?





ECG 83 Analysis: Sinus bradycardia

ECG 83. There is a regular rhythm with a rate of 54 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus bradycardia. Although the PR interval is prolonged (0.22 sec), it may not be a first-degree AV block, as the interval is possibly appropriate for the bradycardia. The PR interval changes with heart rate and become prolonged with sinus bradycardia as a result of decreased sympathetic and increased parasympathetic inputs to the AV node. With sinus tachycardia sympathetic stimulation increases the conduction velocity through the AV node, resulting in a decrease in the PR interval.

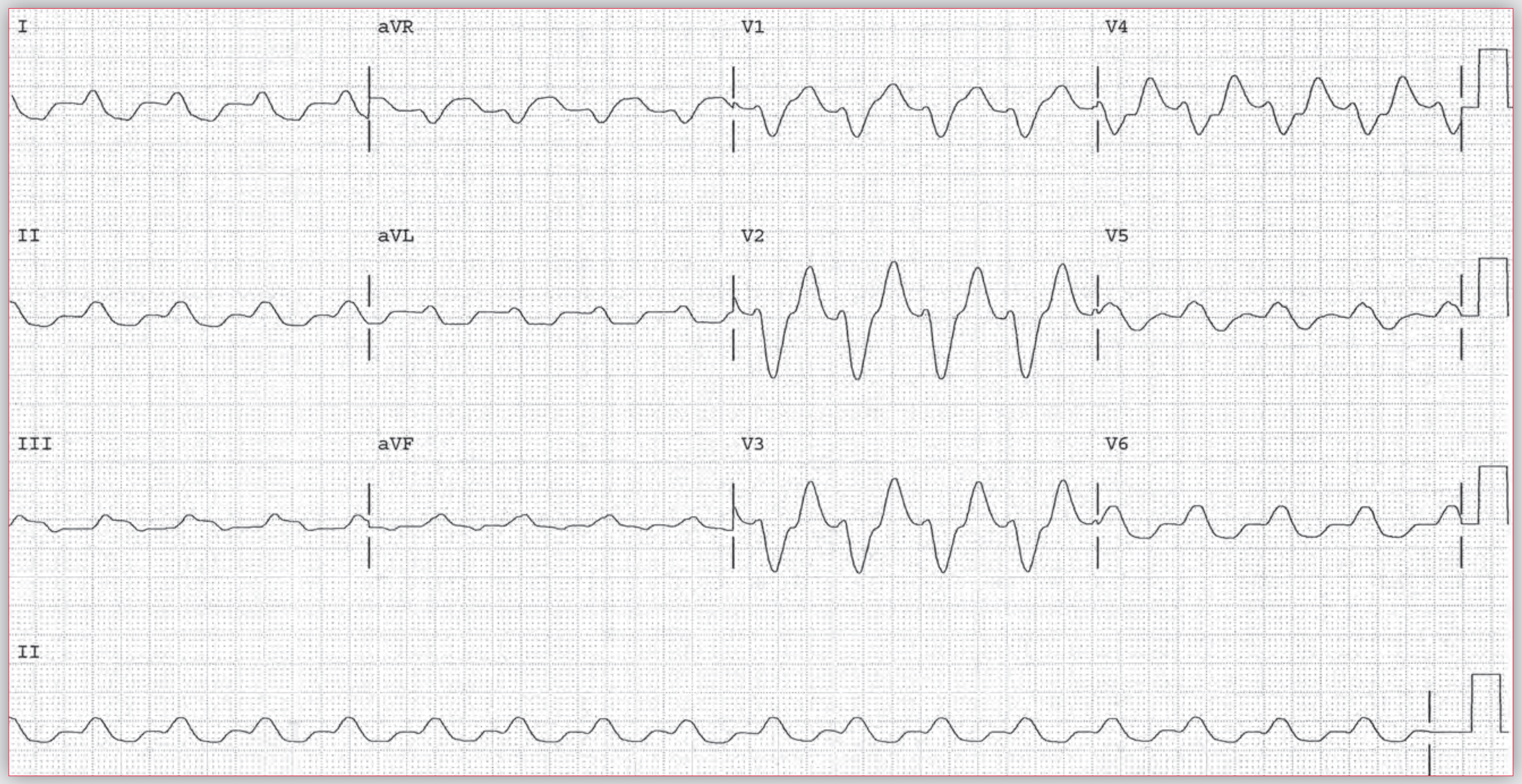
The QRS complex duration is normal (0.08 sec). There is an RSR′ morphology in lead V1 (←), a result of a right ventricular conduction delay. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (460/440 msec). The T waves (↓) are not tall or peak, but they are symmetric in all leads.

The normal T wave is asymmetric, regardless of amplitude. The upstroke of the T wave is slower, while the downstroke of the T wave is faster. T waves that are tall, peaked and more importantly symmetric (upstroke and downstroke equal) are seen with hyperkalemia, which may be systemic or localized as in acute MI. In this case the hyperkalemia is the result of dehydration, renal insufficiency, the use of an ACE inhibitor, acidosis, and the absence of insulin. ■

Core Case 84

An 88-year-old woman is brought to the emergency department because of fever to 104°F associated with shaking chills. Evaluation showed a WBC count of 24,000 and evidence of pyelonephritis. Shortly after presentation, she becomes hypotensive and hypoxic and requires intubation and pressors. It was felt that she was in septic shock. An ECG

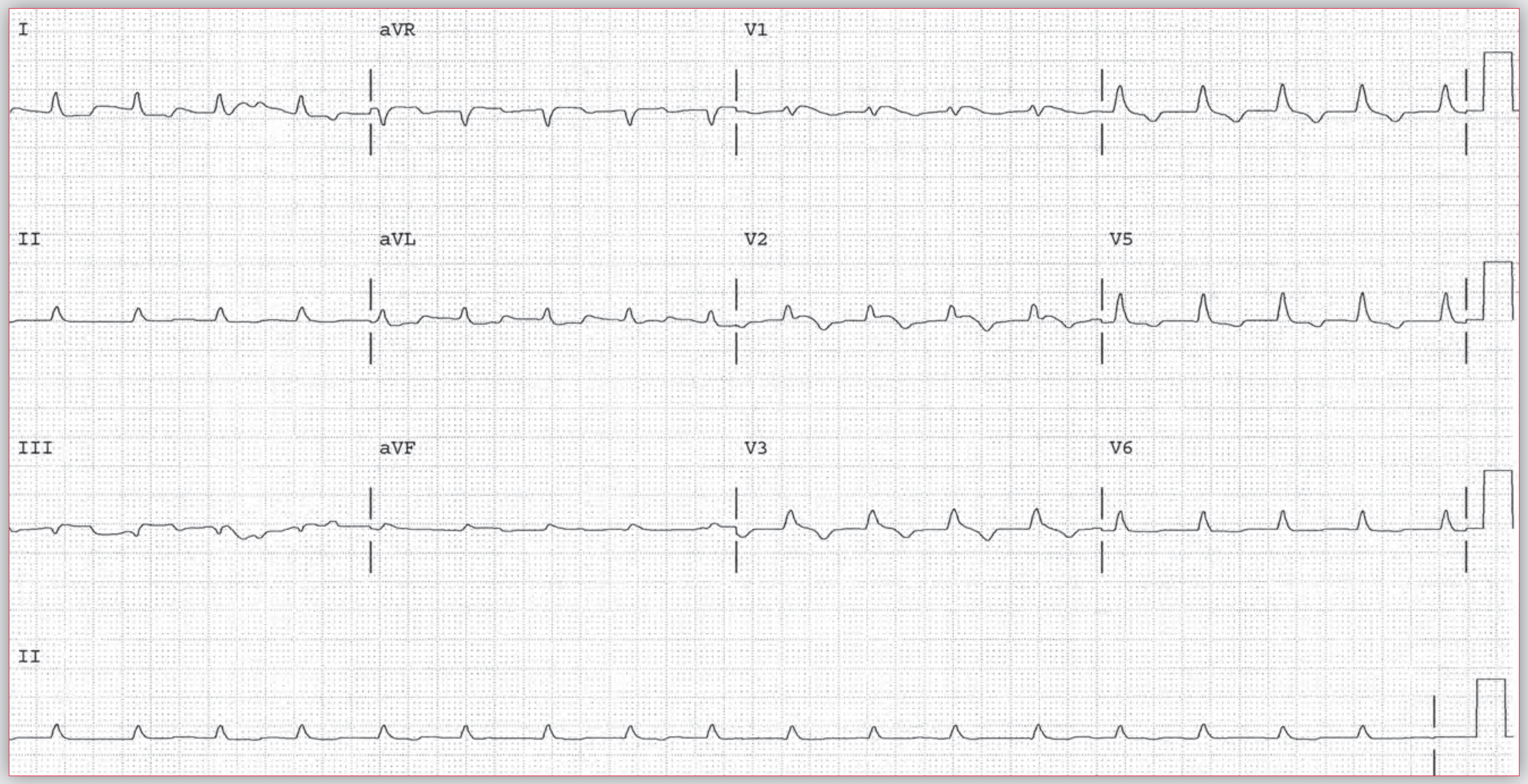
ECG 84A

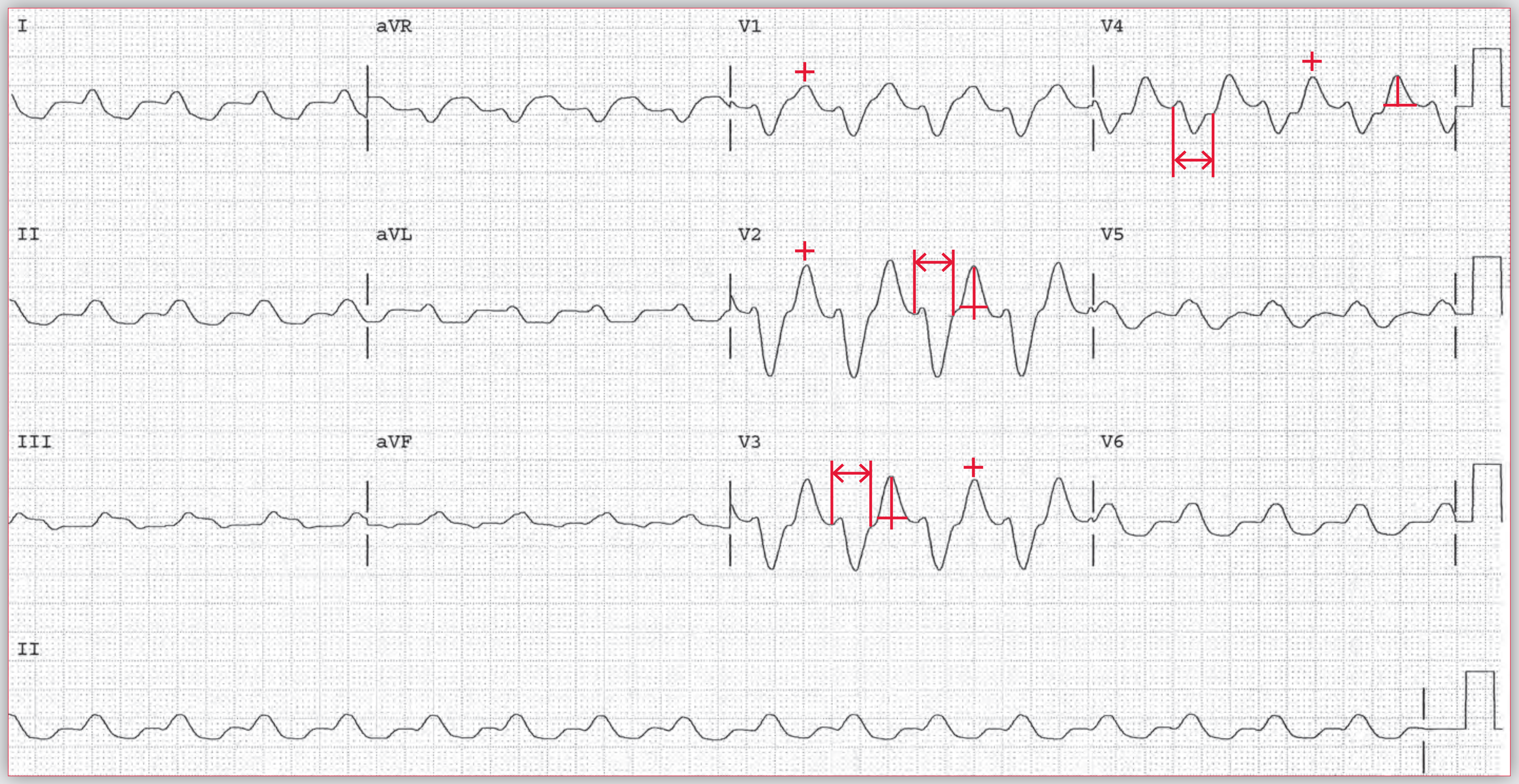


showed sinus tachycardia but was otherwise unremarkable. On the following day, it was noted that she was in acute renal failure on top of chronic renal insufficiency (previous creatinine was 2.2 mg/dL) with a creatinine that was 4.5 mg/dL. An ECG was repeated (ECG 84A). A follow-up ECG (ECG 84B) was obtained after therapy.

What is the most likely diagnosis?

ECG 84B





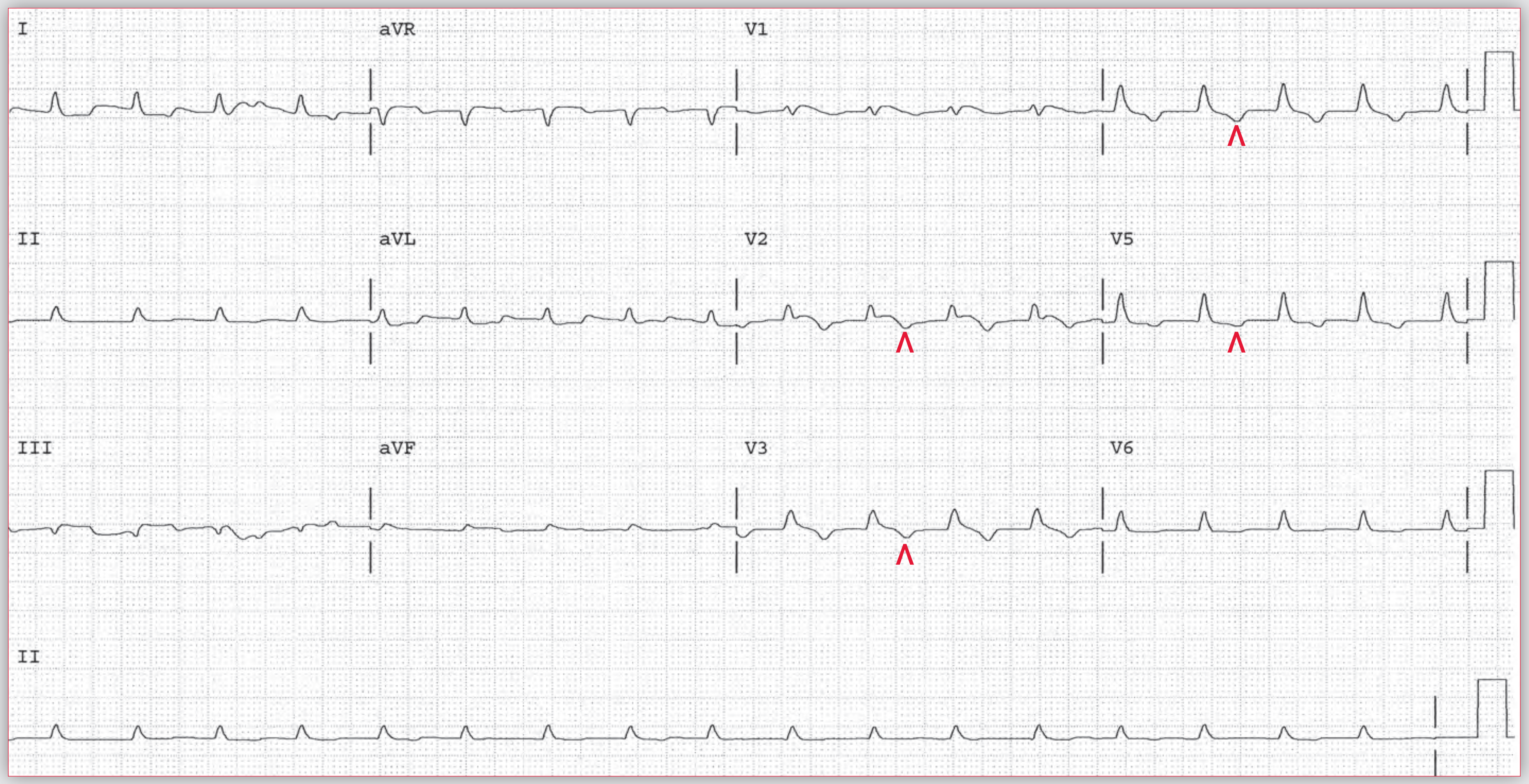
ECG 84A Analysis: Regular rhythm, marked QRS prolongation, hyperacute T waves, hyperkalemia

ECG 84A shows there is a regular rhythm with a rate of 100 bpm. There are no P waves seen before or after any QRS complexes. The QRS complex duration is very prolonged (↔) (0.28 sec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (520/670 msec) but they are normal when the widened QRS complex duration is considered (360/460 msec). The T waves (+) are symmetric, although not tall or peaked.

The only condition that will prolonged the QT ≥ 0.24 sec is hyperkalemia, as a result of diffuse slowing of conduction through the His-Purkinje system. The conduction velocity is determined by phase 0 (upstroke) of the fast action potential that is mediated by rapid influx of sodium ions. The influx rate is related to the relationship between the resting membrane potential (normal = –90 mV) and the threshold potential (–60 mV) after which a spontaneous action potential occurs. The closer the resting membrane potential is to the threshold potential, the less rapid is the influx of sodium ions and the slower is conduction velocity. Membrane threshold potential is determined by the ratio of intracellular and extracellular potassium. Intracellular potassium concentration is much higher than the extracellular concentration. When the extracellular potassium level increases, the resting membrane potential is less negative and hence closer to the threshold potential. Therefore, the influx of sodium ions slows and the conduction velocity decreases, causing a widening of the QRS complex duration.

The atrial myocardium is more sensitive to the effect of hyperkalemia and there is often loss of atrial activation as a result of the slowing of conduction velocity within the atrial myocardium and ultimately failure to activate the atrial myocardium. This produces atrial asystole, *ie*, there is still sinus node activity but no atrial activity, and hence no P wave is seen. This has been termed a sinoventricular rhythm. Atrial asystole may occur before there is marked effect on ventricular myocardial conduction velocity and the QRS complex duration. Although there are no P waves seen, it is not clear if this is a junctional rhythm or actually sinus rhythm with the absence of atrial activation, *ie*, atrial asystole.

continues



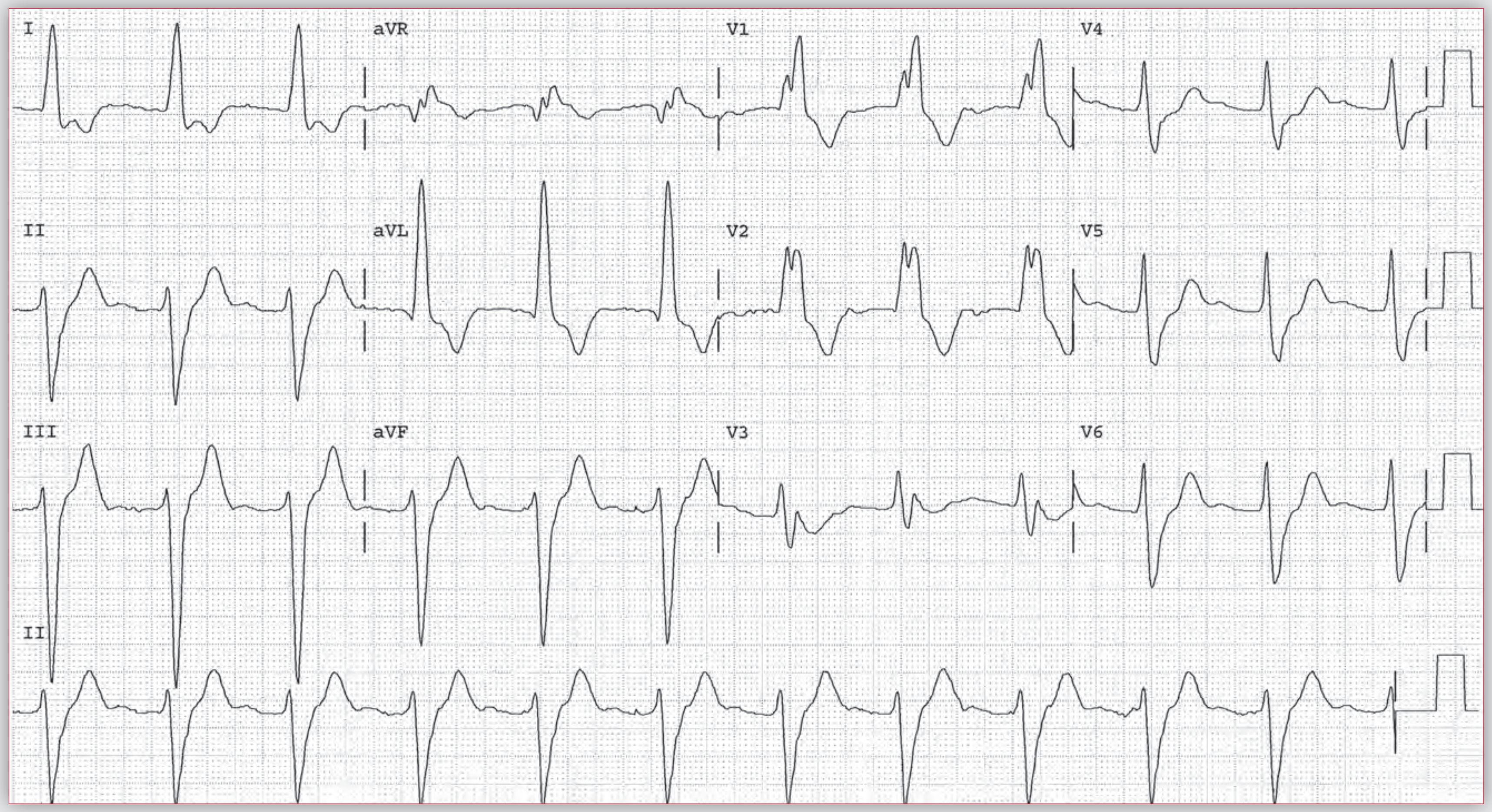
ECG 84B Analysis: Regular narrow complex rhythm (junctional rhythm)

ECG 84B was obtained 30 minutes after therapy for hyperkalemia with glucose, insulin, and kayexelate. The rhythm is regular at a rate of 100 bpm. No P waves are seen before or after any of the QRS complexes. As indicated above, it is not clear if this is a junctional rhythm or actually a sinus rhythm with the absence of atrial activation, *ie*, atrial asystole and a sinoventricular rhythm. The QRS complex duration is 0.10 sec and is narrower and normal. The QRS axis, however, is similar to that seen in ECG 84A. Hence, with a reduction in the serum potassium level, intraventricular conduction improves, and the QRS complex narrows. There are T-wave inversions in leads V3–V6 (^). ■

Core Case 85

A 74-year-old man is undergoing general anesthetic for a total knee replacement. Shortly after administration of succinylcholine, the anesthesiologist notes a change in his QRS morphology on the telemetry. He obtains an ECG (85A) and compares it to the admission ECG (85B).

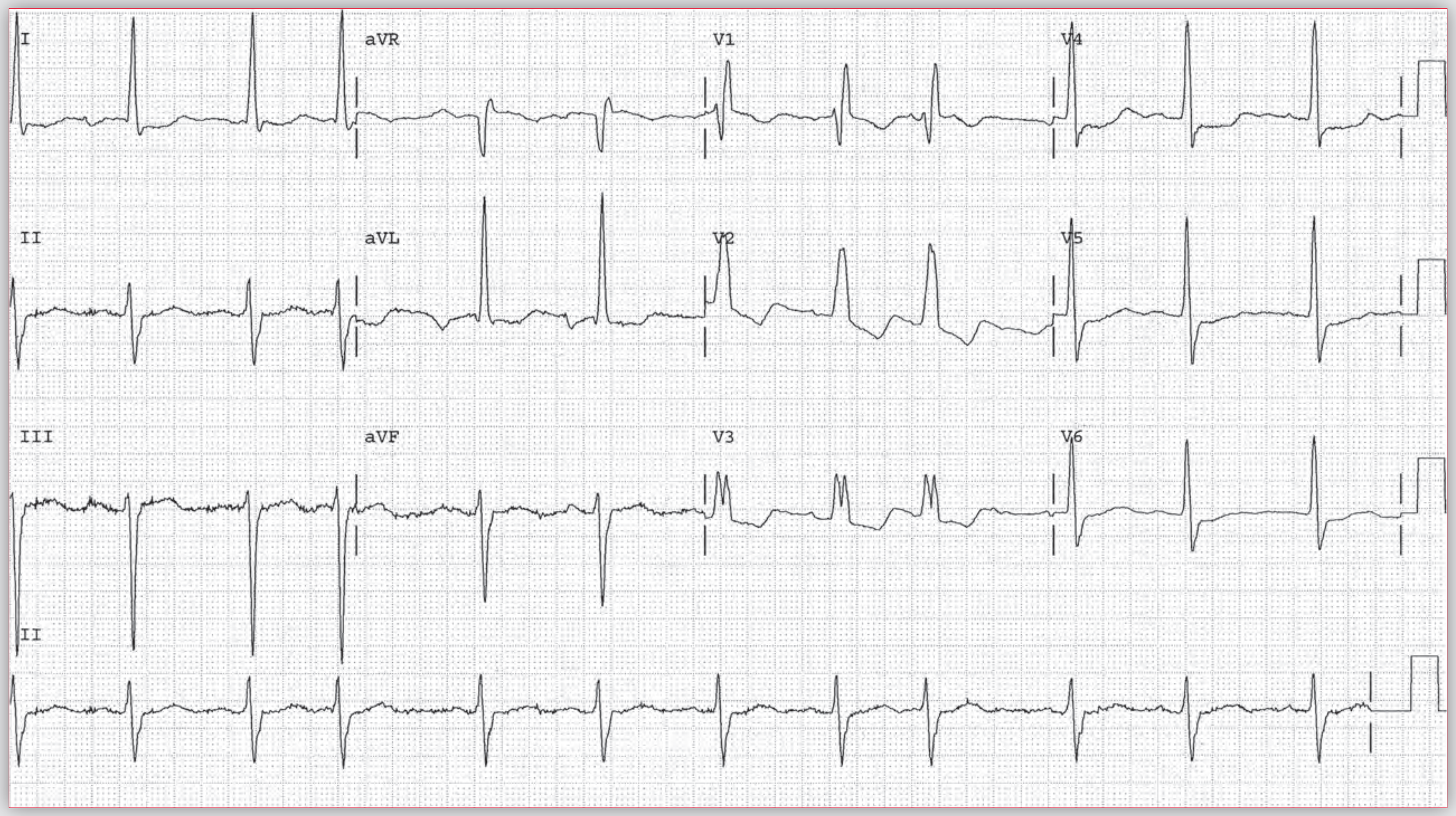
ECG 85A

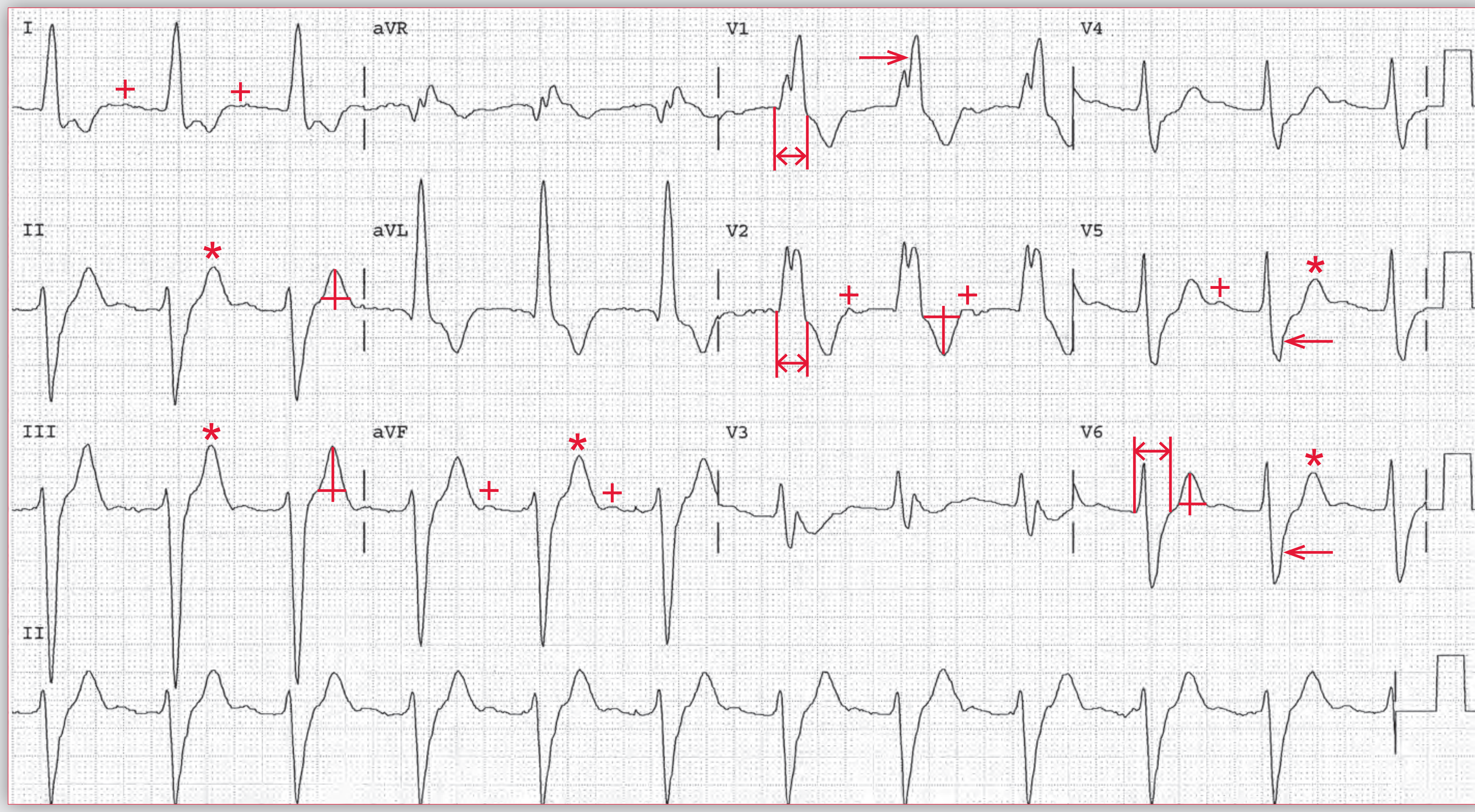


What do the ECGs show?

What is the diagnosis?

ECG 85B

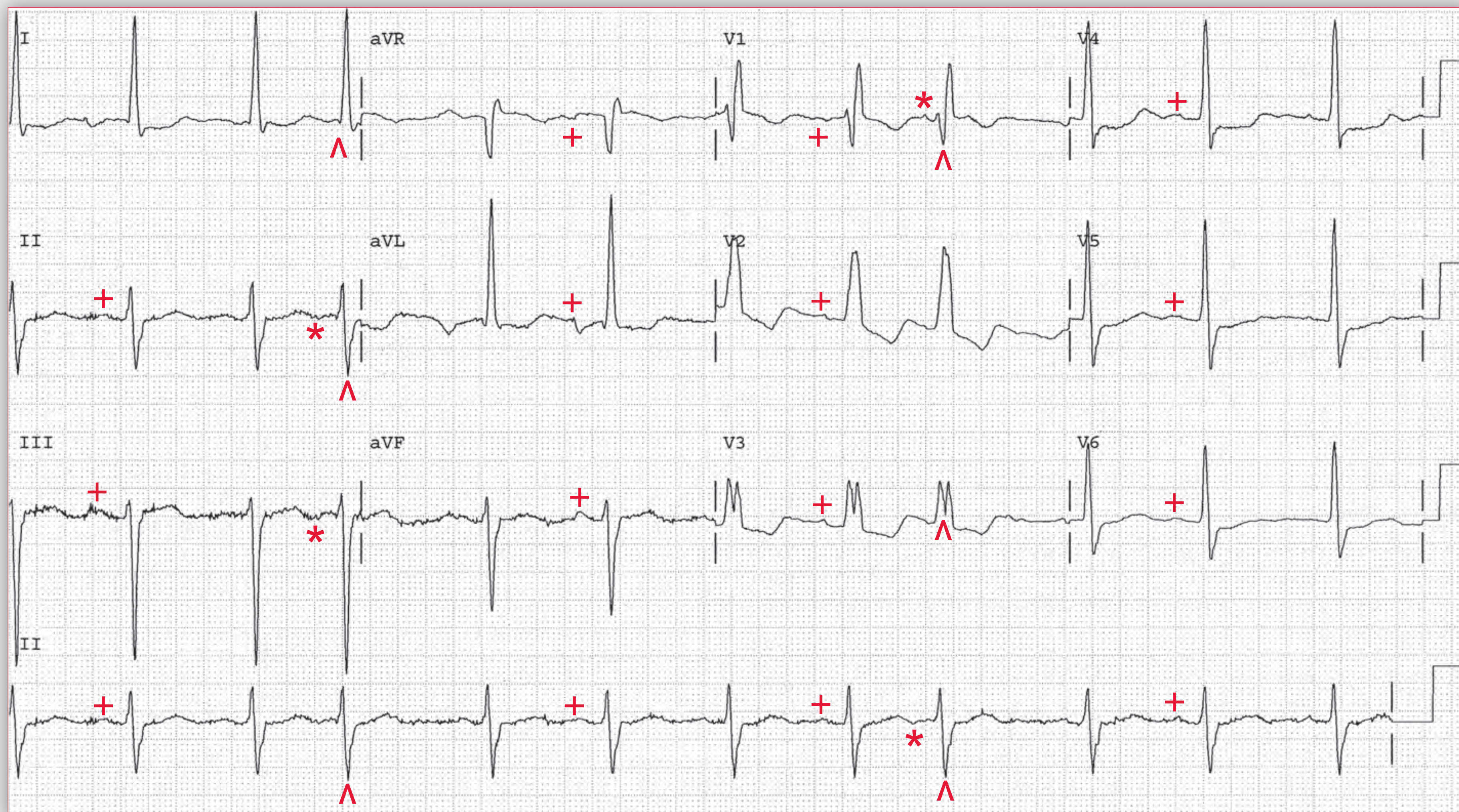




ECG 85A Analysis: Normal sinus rhythm, right bundle branch block (RBBB), left anterior fascicular block (LAFB), marked QRS prolongation and prominent and symmetric T waves, hyperkalemia

ECG 85A shows there is a regular rhythm at a rate of 68 bpm. Although P wave are not seen in most leads, there are P waves (+) seen in leads I, aVF, and V2. There is a stable PR interval (0.36 sec). The QRS complex duration (↔) is prolonged (0.24 sec). The QRS morphology is that of a RBBB with a tall RR′ in V1 (→) and a broad S wave in leads V5–V6 (←). The axis is extremely leftward between −30 and −90 (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology), diagnostic of a LAFB. The QT/QTc intervals are prolonged (500/530 msec), but are normal when the prolonged QRS complex duration is considered (360/380 msec). The QRS width is very prolonged, and the only cause for a QRS duration ≥ 0.24 sec is hyperkalemia. In addition, the T waves (*) are prominent and symmetric. The hyperkalemia is likely the result of succinylcholine therapy.

continues



ECG 85B Analysis: Normal sinus rhythm, first-degree AV block, premature atrial complexes, RBBB, LAFB

ECG 85B is from the same patient as ECG 85A after the correction of hyperkalemia. The rhythm is regularly with a rate of 72 bpm, although the fourth and ninth QRS complexes are premature (^). All the QRS complexes are preceded by a P wave (+) with a stable PR interval (0.26 sec) and the P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block. There is a P wave (*) before each of the premature complexes, although it has a morphology that different than the sinus P waves. Hence these are premature atrial complexes. The QRS complex duration is 0.16 sec and the morphology is a typical RBBB with a LAFB. The QRS complex is similar to the QRS complex in ECG 84A, although the duration is narrower as a result of the decrease in the potassium level. The QT/QTc intervals are the same. ■

Core Case 86

A 55-year-old man with a severe nonischemic cardiomyopathy is treated with furosemide, carvedilol, lisinopril, and spironolactone. One month after starting therapy, he feels very tired and weak. He feels faint and calls his primary care physician,

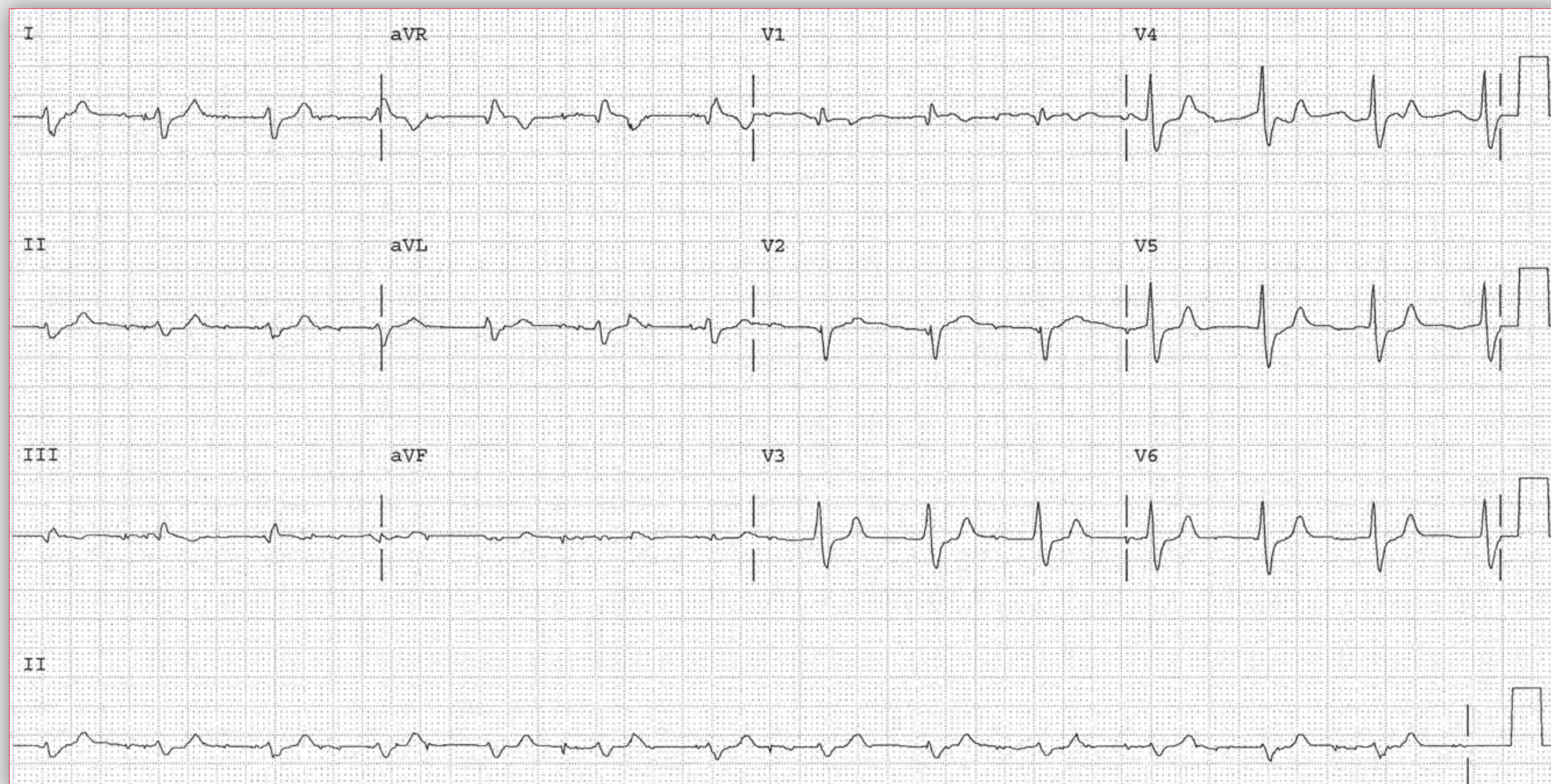
ECG 86A

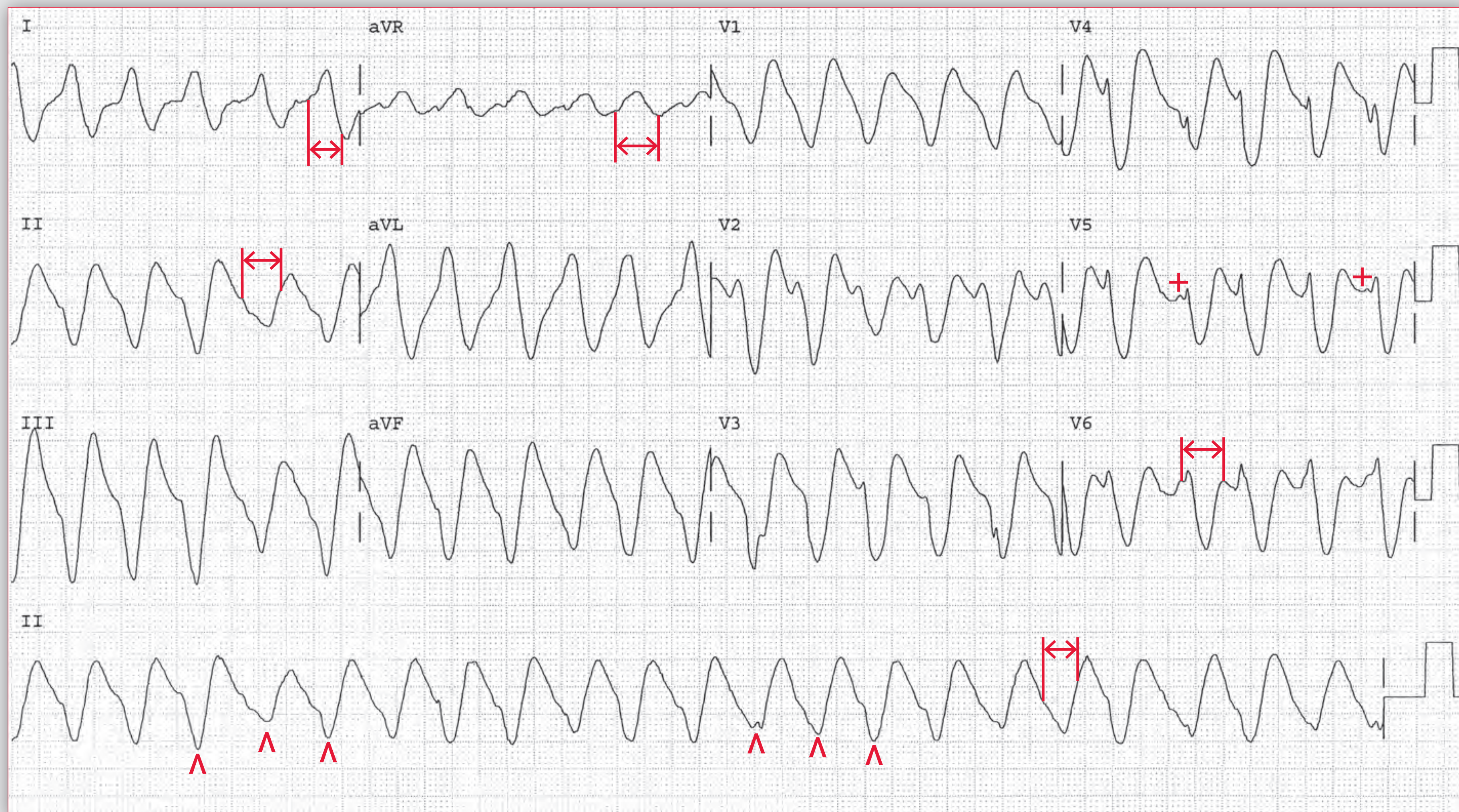
who tells him to come immediately to the emergency department. At presentation, his blood pressure is 70/40 and his mentation is impaired. His initial ECG is shown (ECG 86A). After therapy an ECG is repeated (ECG 86B).

What does it show?

What is the likely diagnosis?

ECG 86B





ECG 86A Analysis: Ventricular tachycardia, hyperkalemia

ECG 86A shows the rhythm is slightly irregular with a rate of 140 bpm. There are no definitive P waves seen before or after any QRS complex, although there are waveforms that may represent atrial activity (**+**), particularly in leads V4–V6. The QRS complex morphology is variable (^) and the duration is prolonged (↔) and also variable (0.28–0.32 sec). The variability in QRS morphology is characteristic of ventricular tachycardia. The markedly prolonged QRS complex duration is seen with hyperkalemia; no other condition, including ventricular tachycardia, prolongs the QRS duration to this extent (*ie*, ≥ 0.24 sec).

Hyperkalemia often occurs in the presence of hemodynamically significant ventricular tachycardia. This is the result of reduced tissue perfusion, hypoxemia, and acidosis. The ventricular tachycardia cannot be reverted until the metabolic abnormalities are corrected. Hence hyperkalemia and acidosis should be treated before attempting to revert the ventricular tachycardia, particularly electrical reversion or cardioversion. Cardioversion in the presence of hyperkalemia has a high potential to result in asystole.

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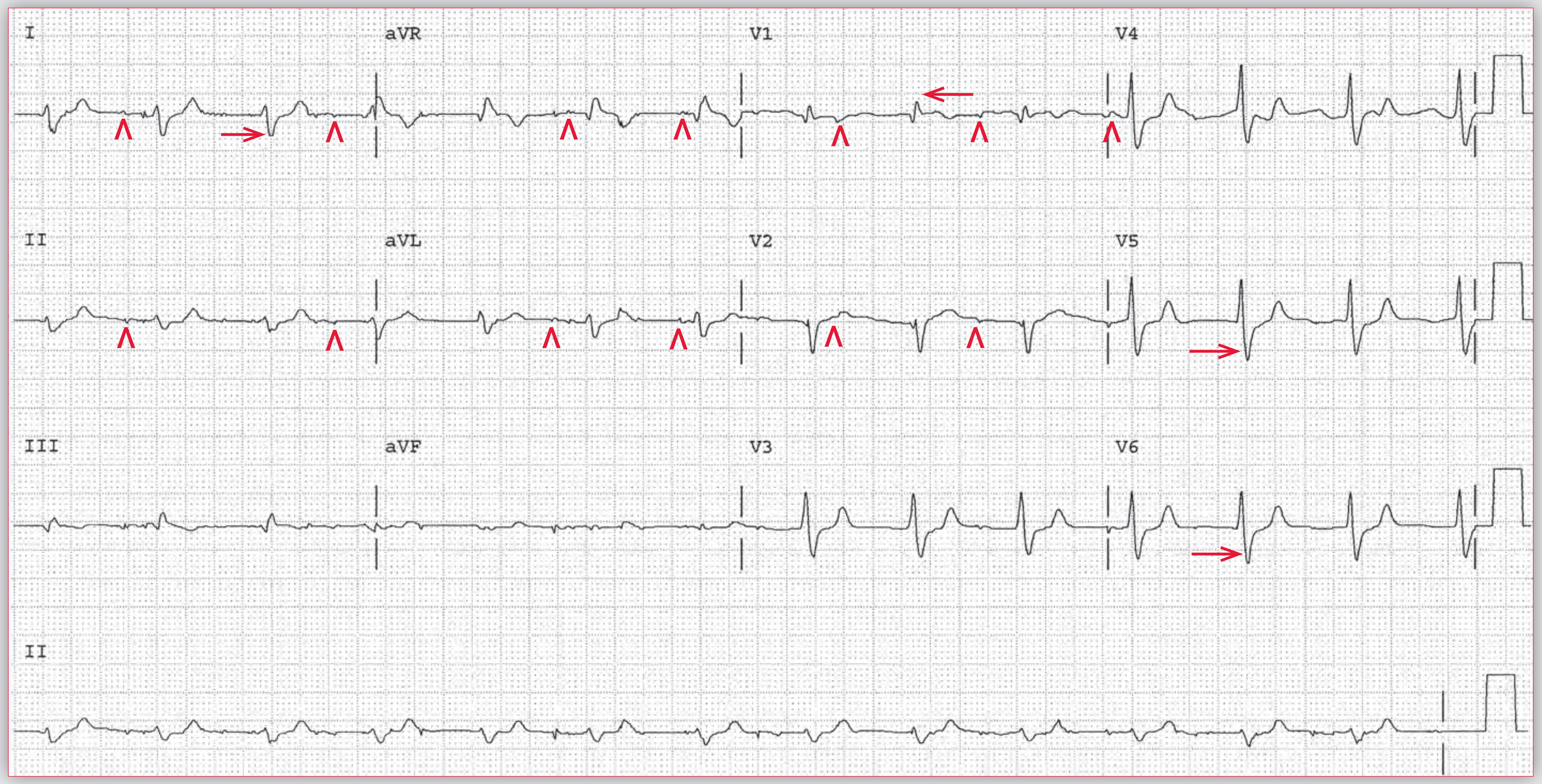


ECG 86B Analysis: Sinus rhythm, AV dissociation, accelerated junctional rhythm, right bundle branch block

ECG 86B was obtained 15 minutes after treatment of hyperkalemia and acidosis. The ventricular tachycardia was reverted with electric cardioversion. The rhythm is regular at a rate of 80 bpm. Small and very narrow P wave can be seen (^). They are dissociated from the QRS complexes and have a slightly irregular PP interval, with a rate of about 60 bpm. As the atrial rate is slower than the ventricular rate, this is an accelerated junctional rhythm.

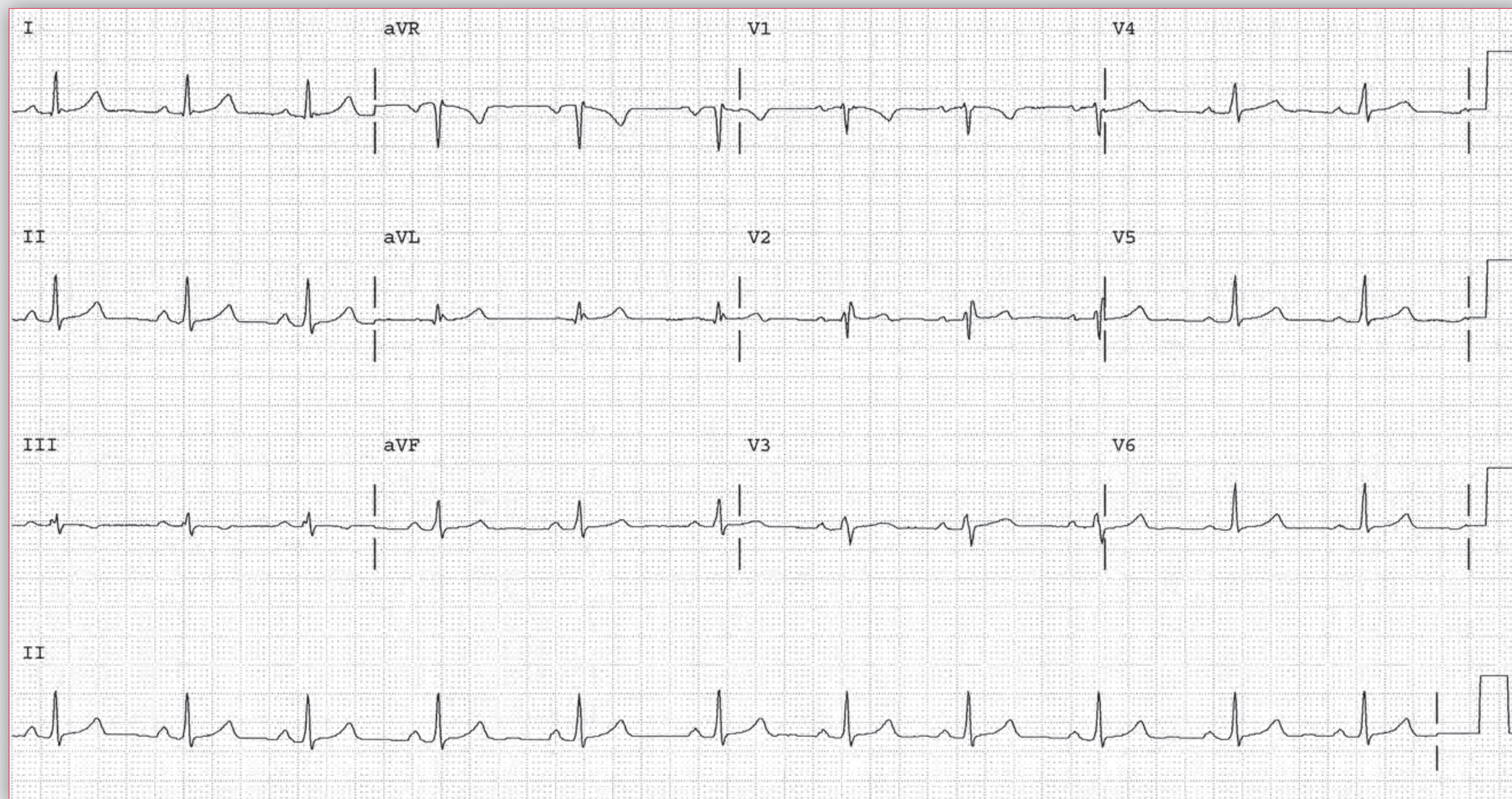
The QRS complex duration is prolonged (0.12 sec) and there is a morphology of a right bundle branch block with a broad R wave in V1 (←) and broad S wave in leads I and V5–V6) (→). There are non-specific ST-T wave changes and the T waves (+) are still symmetric in morphology.

The atrial myocardium is more sensitive to the effect of hyperkalemia, and there is often loss of atrial activation prior to changes of conduction in the ventricular myocardium as a result of the slowing of conduction velocity with the atrial myocardium. This produces atrial asystole, *ie*, there is still sinus node activity but not atrial activity or activation and hence no P wave is seen. With therapy of hyperkalemia, there is a resumption of atrial activity, although it may remain abnormal with small and abnormal P waves. There is AV dissociation presence, but it is not clear if this is the result of hyperkalemia, acidosis, or possibly as a post-ventricular tachycardia condition. ■

Core Case 87

A 36-year-old man with a history of intermittent palpitations sees his primary care physician. He has no other medical problems and is not on any medication. An ECG is obtained (ECG 87A), although he was not

ECG 87A

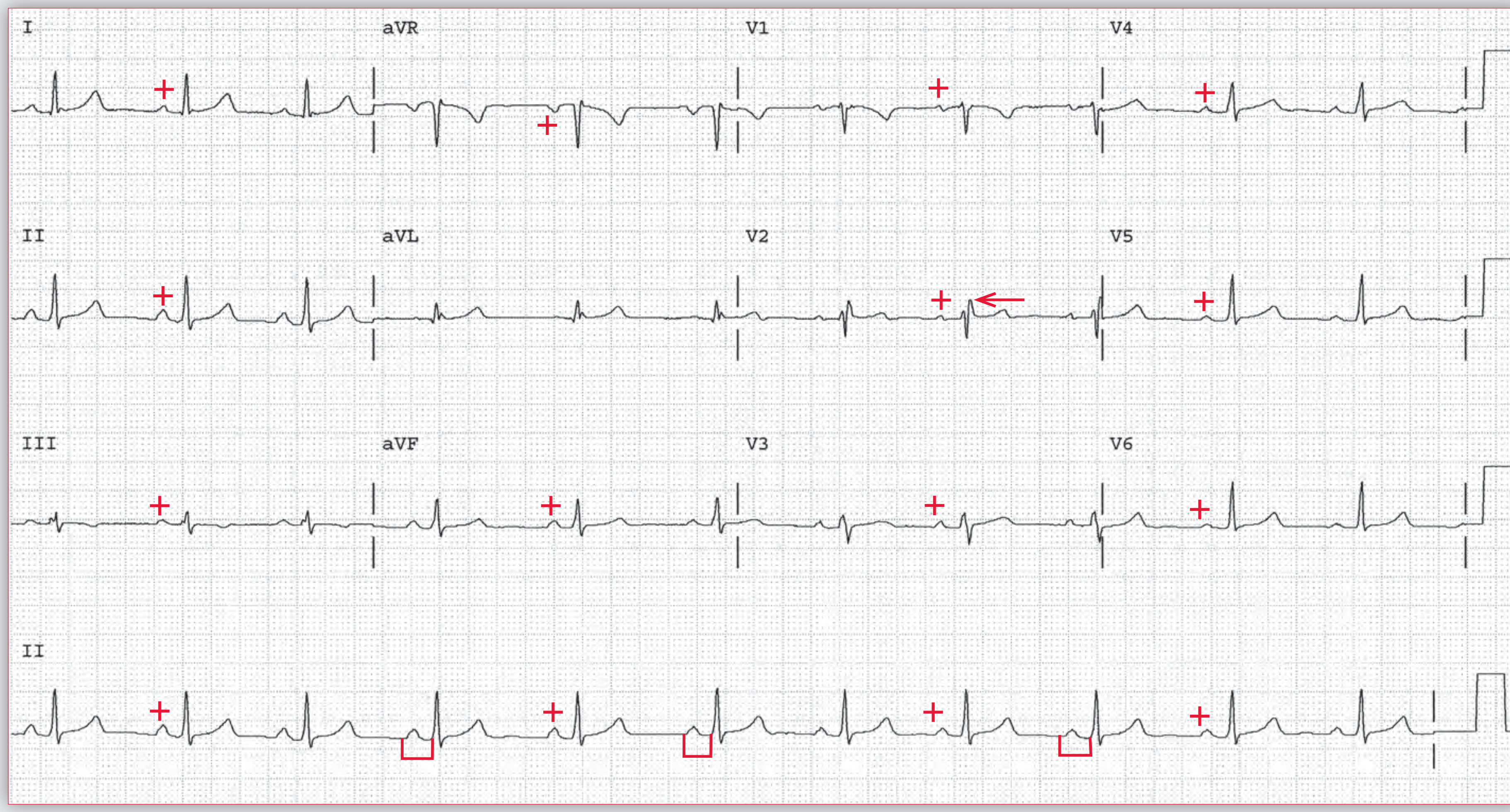


experiencing palpitations at the time. Several minutes later, he again complains of palpitations and the ECG machine is reattached. However, by the time the ECG is recorded (ECG 87B), the palpitations have resolved.

What is the difference between the two ECGs?

Is there a clue to the etiology of the palpitations based on these ECGs?

ECG 87B



ECG 87A Analysis: Normal sinus rhythm, right ventricular conduction delay, low voltage in the precordial leads

ECG 87A shows there is a regular rhythm at a rate of 66 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (⊔) (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal, although there is a RSR′ morphology in V2 (←), representing a right ventricular conduction delay. The voltage in the precordial leads (V1–V6) is low, *ie*, the amplitude is less than 10 mm (boxes) in each lead. The QT/QTc intervals are normal (400/420 msec).

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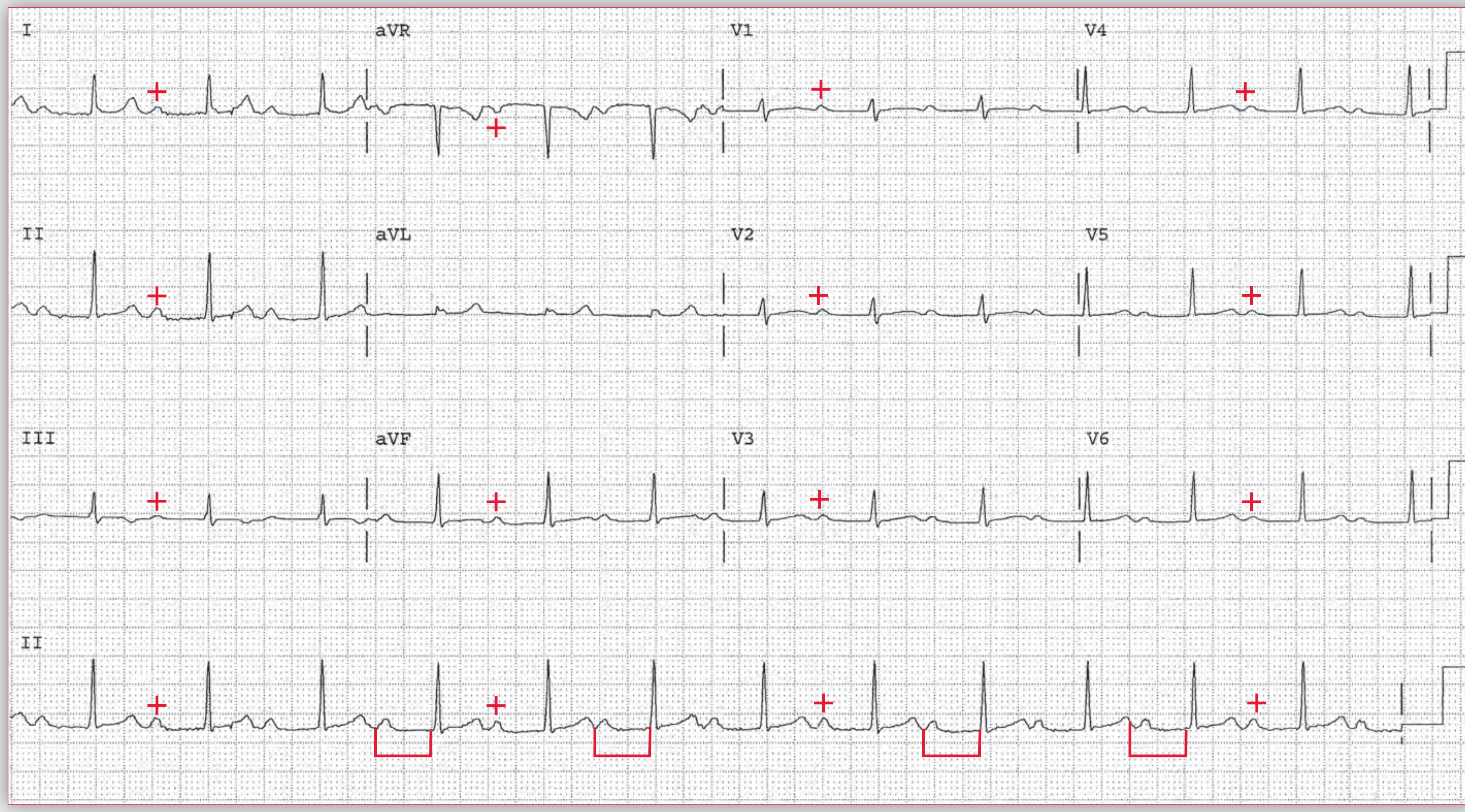


ECG 87B Analysis: Normal sinus rhythm with marked first-degree AV block, dual AV nodal pathways

ECG 87B was obtained from the same patient in ECG 87A about 30 minutes later. The patient did not receive any medication. The rate is 76 bpm and there is a P wave (+) before each QRS complex. The P wave and QRS morphology, QRS duration and axis, and QT/QTc intervals are the same as in ECG 87A. However, the PR interval is much longer (⊔) (0.40 sec) and constant. Therefore, the patient has two different PR intervals, one shorter and the other longer. This is the manifestation of dual AV nodal pathways, one of which conducts slowly but has a short refractory period, *ie*, recovers more quickly, while the other conducts more rapidly but has a long refractory period, *ie*, takes longer to recover. The two pathways are linked via the atrial myocardium proximally and the bundle of His distally.

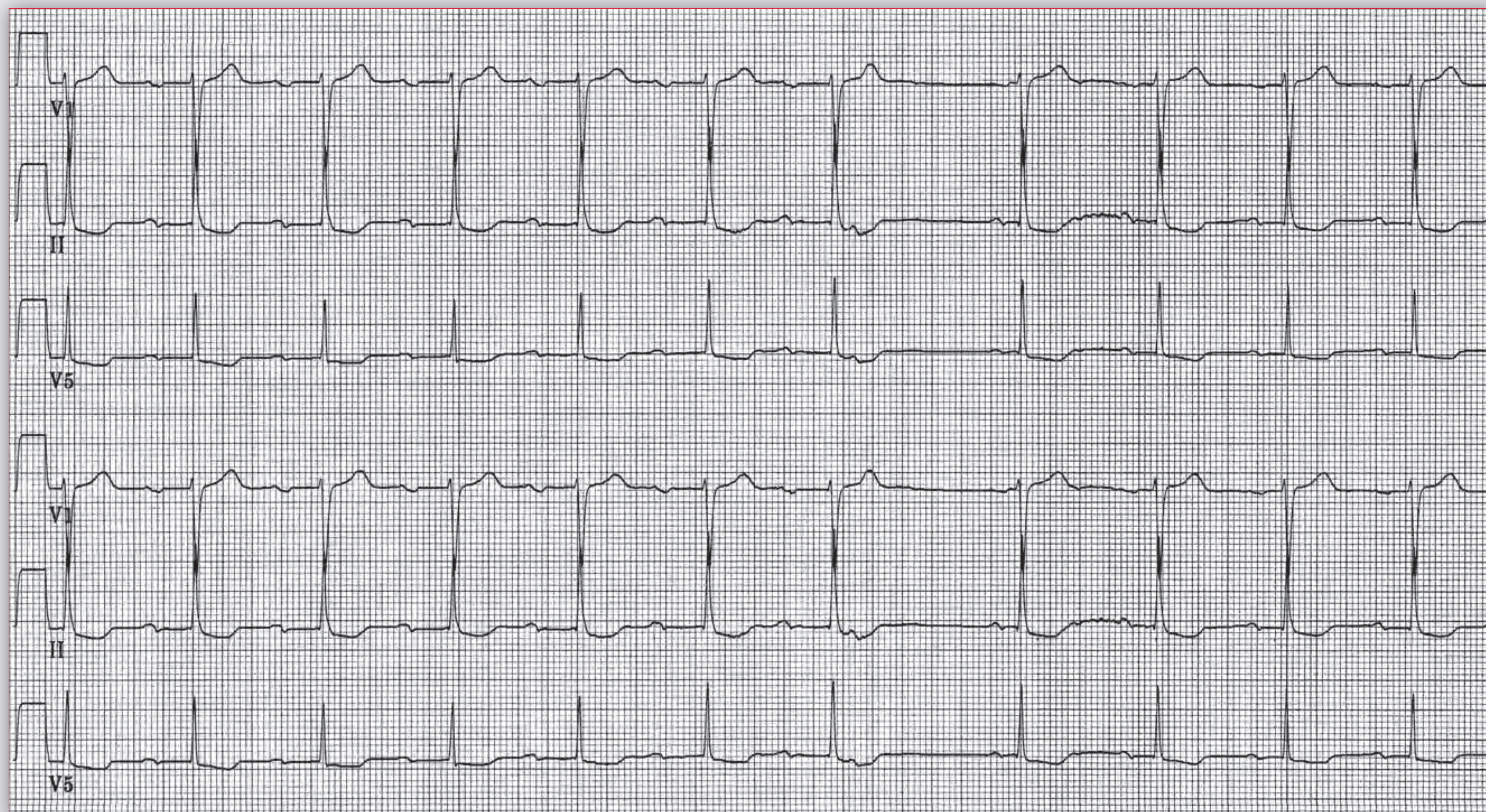
The presence of dual AV nodal pathways is the anatomic etiology for the occurrence of an atrioventricular nodal reentrant tachycardia (AVNRT). Under normal conditions, the activation of the ventricles is via impulse conduction through the fast pathway that predominates. However, if there is a premature atrial impulse that arrives at the AV node before the fast pathway has recovered, the impulse can be conducted through the slow pathway if it has already recovered. The premature atrial complex will have a long PR interval. If the impulse arrives at the distal connection of the two pathways (*ie*, bundle of His) at a time when the fast pathway has recovered, the impulse will be conducted retrogradely back to the atria via the fast pathway at the same time that the ventricles are activated antegradely via the His-Purkinje system. If the retrograde impulse arrives at the proximal connection of the two pathways when the slow pathway has recovered, it can reenter this pathway and the process then continues, resulting in the reentrant arrhythmia known as AVNRT. In this situation, there is simultaneous activation of the ventricles (antegradely) and the atria (retrogradely). Hence there is no obvious P wave seen, *ie*, this is a no-RP tachycardia. ■

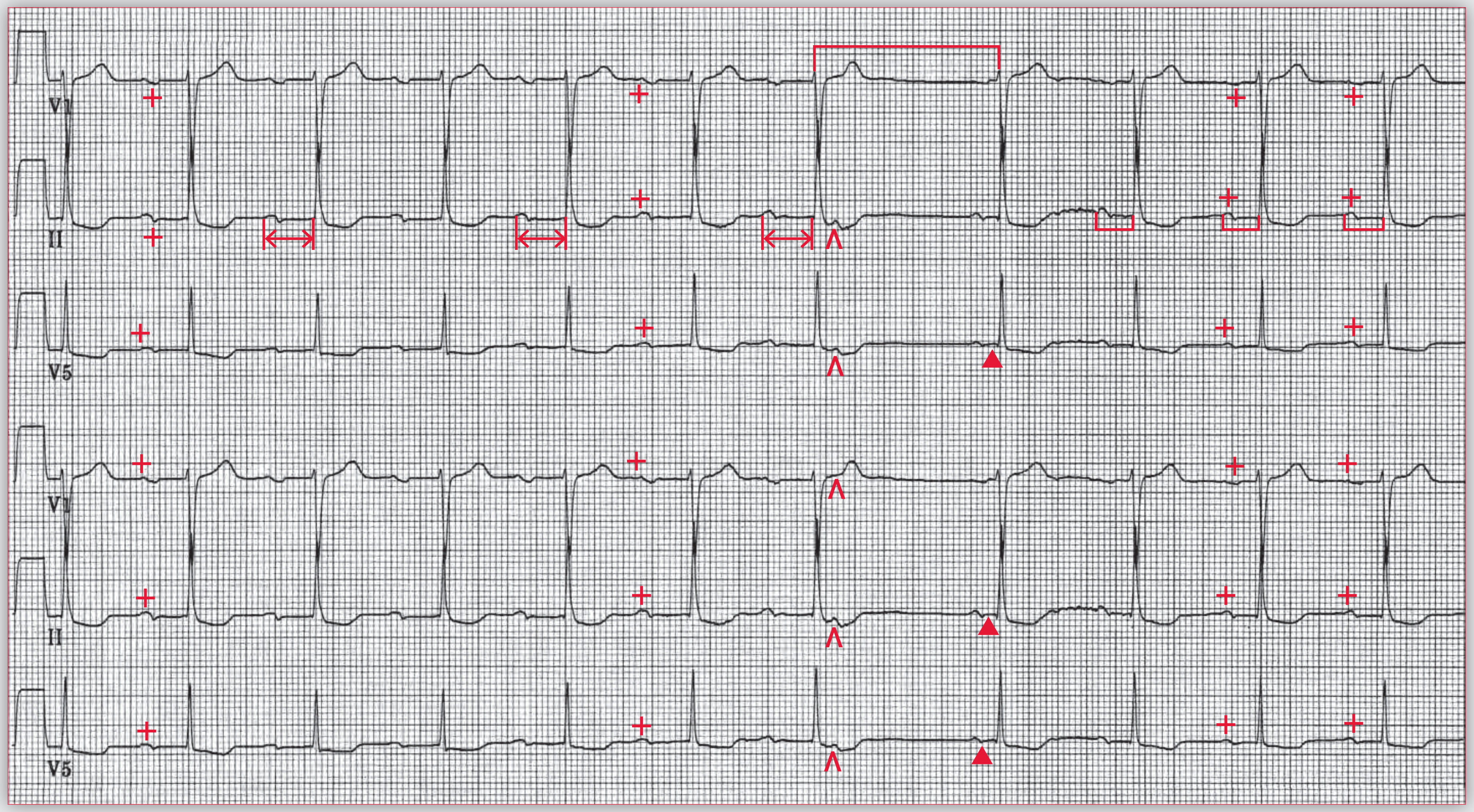
Notes

Core Case 88

A 24-year-old woman with palpitations is seen by her primary care physician. Though she does not have palpitations at the time of this rhythm strip, it does suggest an etiology of her palpitations.

What is the most likely explanation?





ECG 88 Analysis: Normal sinus rhythm, blocked premature atrial contraction, junctional escape complex, dual AV nodal pathways

There are three rhythm strips. There is a regular rhythm at a rate of 66 bpm. The QRS complex duration is normal (0.08 sec) and the QT/QTc intervals are normal (380/400 msec). There is a P wave (+) before each QRS complex. In the initial part of the rhythm strip, there is a stable PR interval (↔) that is 0.36 sec. There is a pause after the seventh QRS complex (⊓), which is due to a nonconducted premature P wave (^), observed in the early part of the ST segment. This is a nonconducted premature atrial premature complex. After the pause, there is resumption of a stable sinus rhythm at the same rate as before the pause and with the same P wave morphology (+). However, the PR interval is shorter (0.24 sec) (⊔). This is a result of dual AV nodal pathways, *ie*, a slow-conducting pathway accounting for the longer PR interval and a fast-conducting pathway accounting for the shorter PR interval. The PR interval of the complex after the pause (▲) is slightly shorter than the other PR intervals (*ie*, 0.20 sec vs. 0.24 sec). This may be the result of faster AV conduction after the pause or due to an escape junctional or nodal complex that results from the long RR interval.

When there are dual AV nodal pathways, one conducts slowly but has a short refractory period, *ie*, recovers more quickly, and the other conducts more rapidly but has a long refractory period, *ie*, takes longer to recover. The two pathways are linked via the atrial myocardium proximally and the bundle of His distally.

The presence of dual AV nodal pathways is the anatomic etiology for the occurrence of an atrioventricular nodal reentrant tachycardia (AVNRT). Under normal conditions, the activation of the ventricles is via impulse conduction through the fast pathway that predominates. However, if there is a premature atrial impulse that arrives at the AV node before the fast pathway has recovered, the impulse can be conducted through the slow pathway if it has already recovered. The premature atrial complex will have a long PR interval. If the impulse arrives at the distal connection of the two pathways (*ie*, bundle of His) at a time when the fast pathway has recovered, the impulse will be conducted retrogradely back to the atria via the fast pathway at the same time that the ventricles are activated antegradely via the His-Purkinje system. If the retrograde impulse arrives at the proximal connection of the two pathways when the slow pathway has recovered, it can reenter this pathway and the process then continues, resulting in AVNRT. In this situation, there is simultaneous activation of the ventricles (antegradely) and the atria (retrogradely). Hence there is no obvious P wave seen, *ie*, this is a no-RP tachycardia. ■

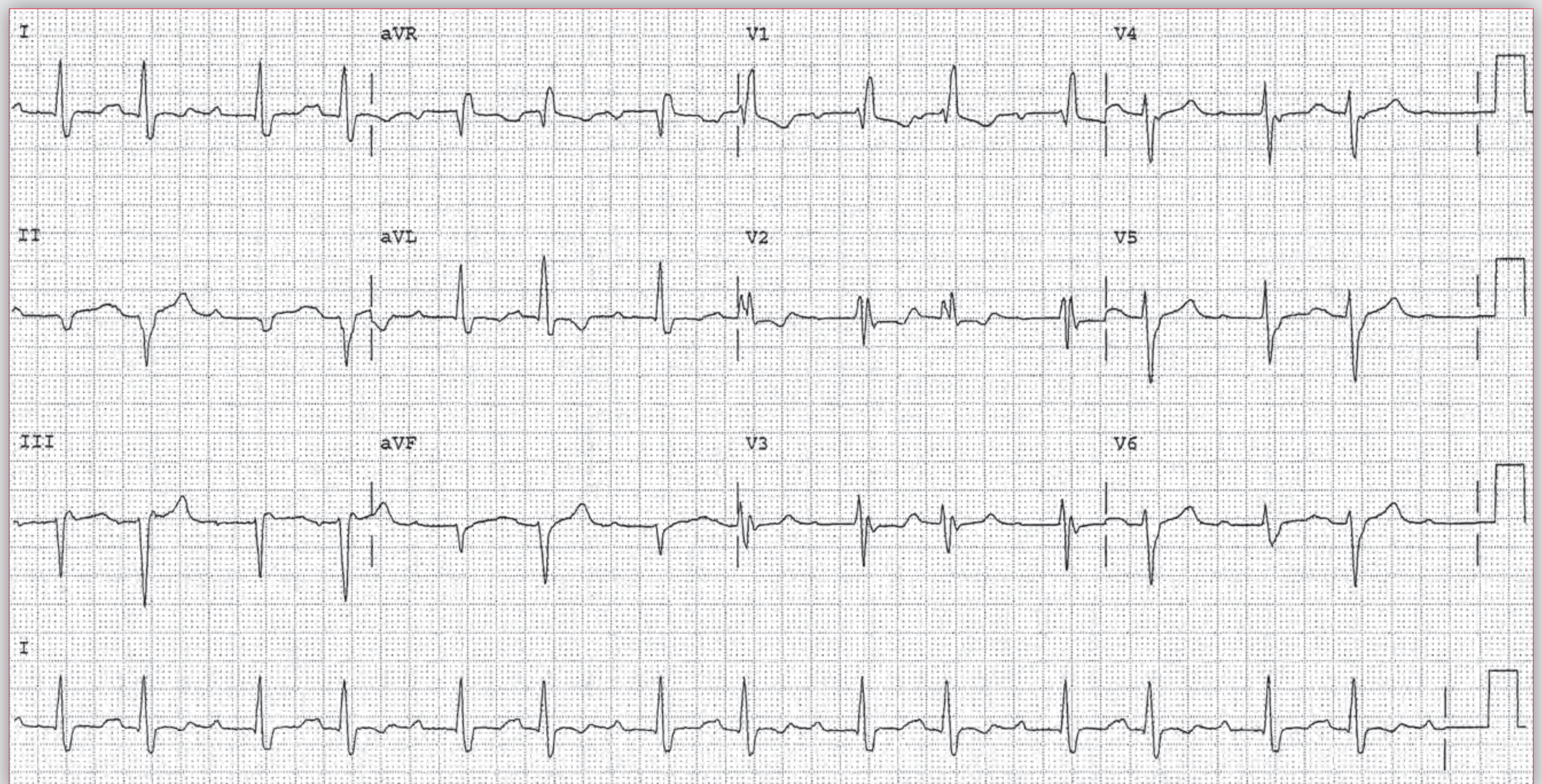
Notes

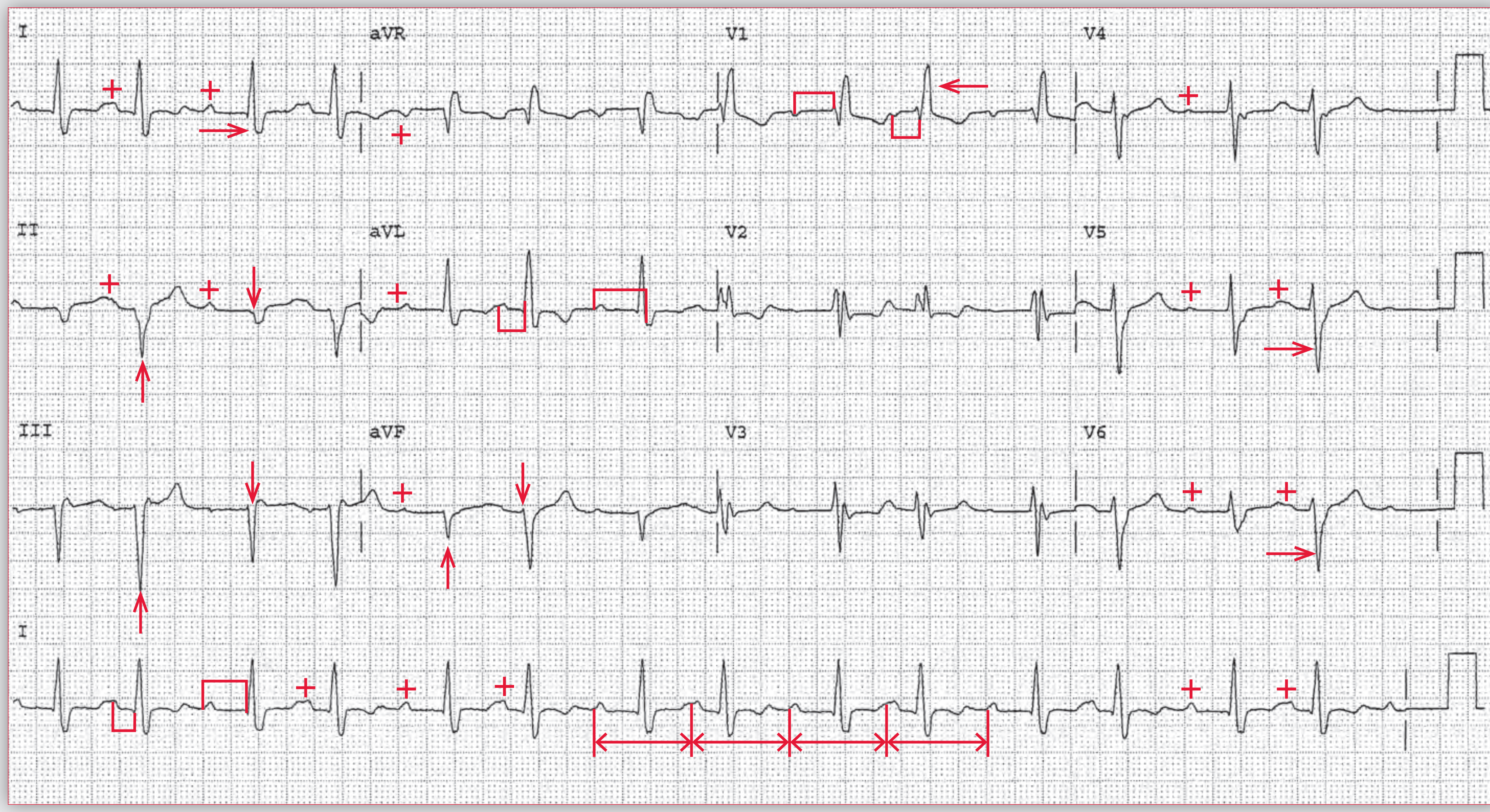
Core Case 89

A 35-year-old woman is seen for a routine physical examination before undergoing a D and C for excessive vaginal bleeding. She has no major medical problems, although she does state that she has had a long history of palpitations that have lasted for up to 3 hours. The episodes are often terminated by coughing. She has never had an ECG taken during an episode. An ECG is obtained.

What does this show?

Does the finding suggest an etiology for her palpitations?





ECG 89 Analysis: Normal sinus rhythm, right bundle branch block, left anterior fascicular block, dual AV nodal pathways

There is a regularly irregular rhythm with an average rate of 84 bpm. There is a P wave (+) before each QRS complex with a regular rate of 84 bpm (↔). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The PR intervals are not constant, and there is a repeating pattern of long (⊓) and short (⊔) PR intervals. The long PR interval is 0.34 sec while the shorter PR interval is 0.22 sec. The regularly irregular RR intervals are the result of the two different PR intervals as the PP intervals are regular (↔).

The QRS complex duration is increased (0.14 sec) and the morphology is typical for a right bundle branch block with an RSR′ in lead V1 (←) and a broad S wave in leads I and V5–V6 (→). The axis is extremely leftward between –30° and –90° (positive QRS complex in lead I and negative in leads II and aVF with an rS morphology). This is a left anterior fascicular block. Therefore, there is bifascicular block present. The QT/QTc intervals are prolonged (420/500 msec) but are normal when the prolonged QRS complex duration is considered (360/430 msec).

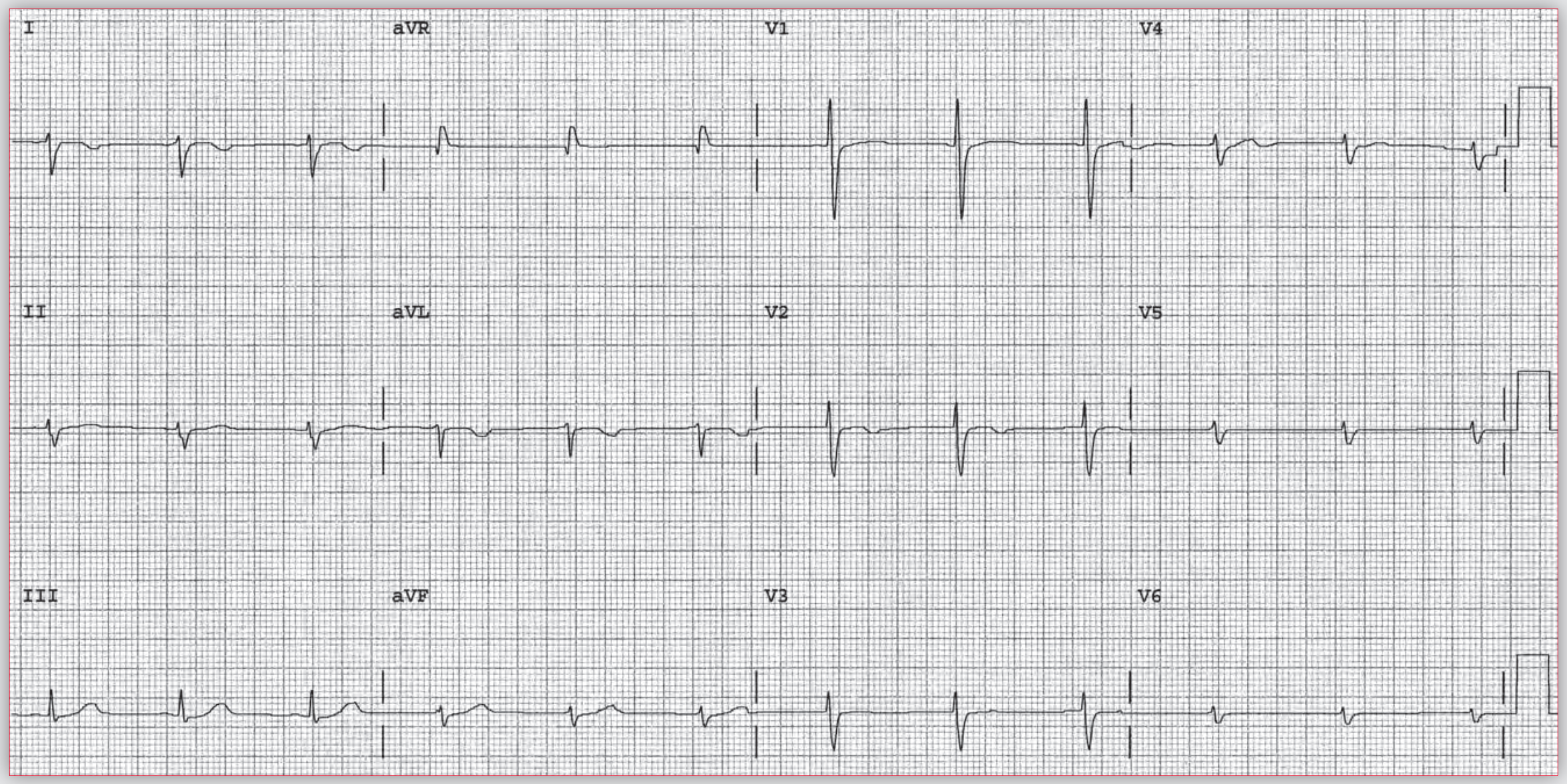
The presence of two distinct PR intervals that alternate in a repeating pattern in the presence of a regular sinus rhythm means that there is alternating or beat-to-beat changes in AV nodal conduction. The only etiology for this are dual AV nodal pathways, one that has fast conduction (but a long refractory period or slow recovery) and the other slow conducting (but a short refractory period or fast recovery). These two pathways are linked proximally in the atrial myocardium and distally in the bundle of His. The presence of dual AV nodal pathways is a precondition for the development of atrioventricular nodal reentrant tachycardia (AVNRT). When there is a premature atrial complex that reaches the fast pathway before it recovers, the impulse is conducted to the ventricle via the slow pathway. If it reaches the distal portion of the fast pathway at a time when it has recovered, it can enter the fast pathway and conduct retrogradely to the atrium at the same time the impulse is conducted antegradely to the ventricle via the His-Purkinje system. If the retrogradely conducted impulse reaches the proximal portion of the slow pathway after it has recovered, it can reenter this pathway and if this pattern repeats, a reentrant arrhythmia is established, *ie*, AVNRT.

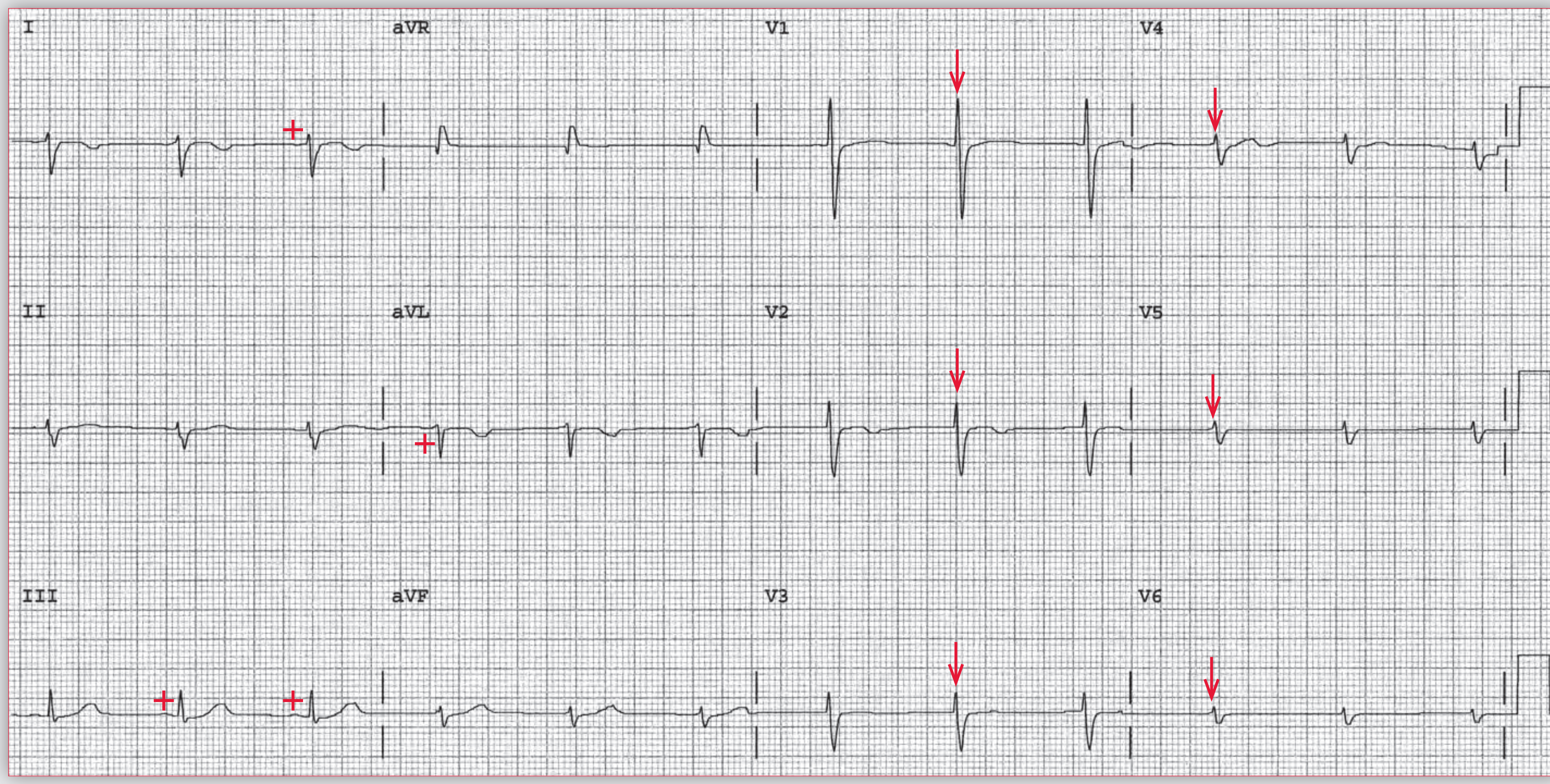
In addition, there are beat-to-beat differences in the amplitude of the two QRS complexes (↓, ↑). This is not electrical or QRS complex alternans as the RR intervals are irregular. The changes in amplitude are due to the fact that the two different AV nodal pathways enter the bundle of His at two different locations and hence are conducted through the His-Purkinje system via two different pathways or tracks, resulting in slightly different activation sequences within the left ventricle. ■

Notes

A 32-year-old man is seen in the office for a pre-employment physical exam. His ECG is shown.

What is the next test the physician should order?





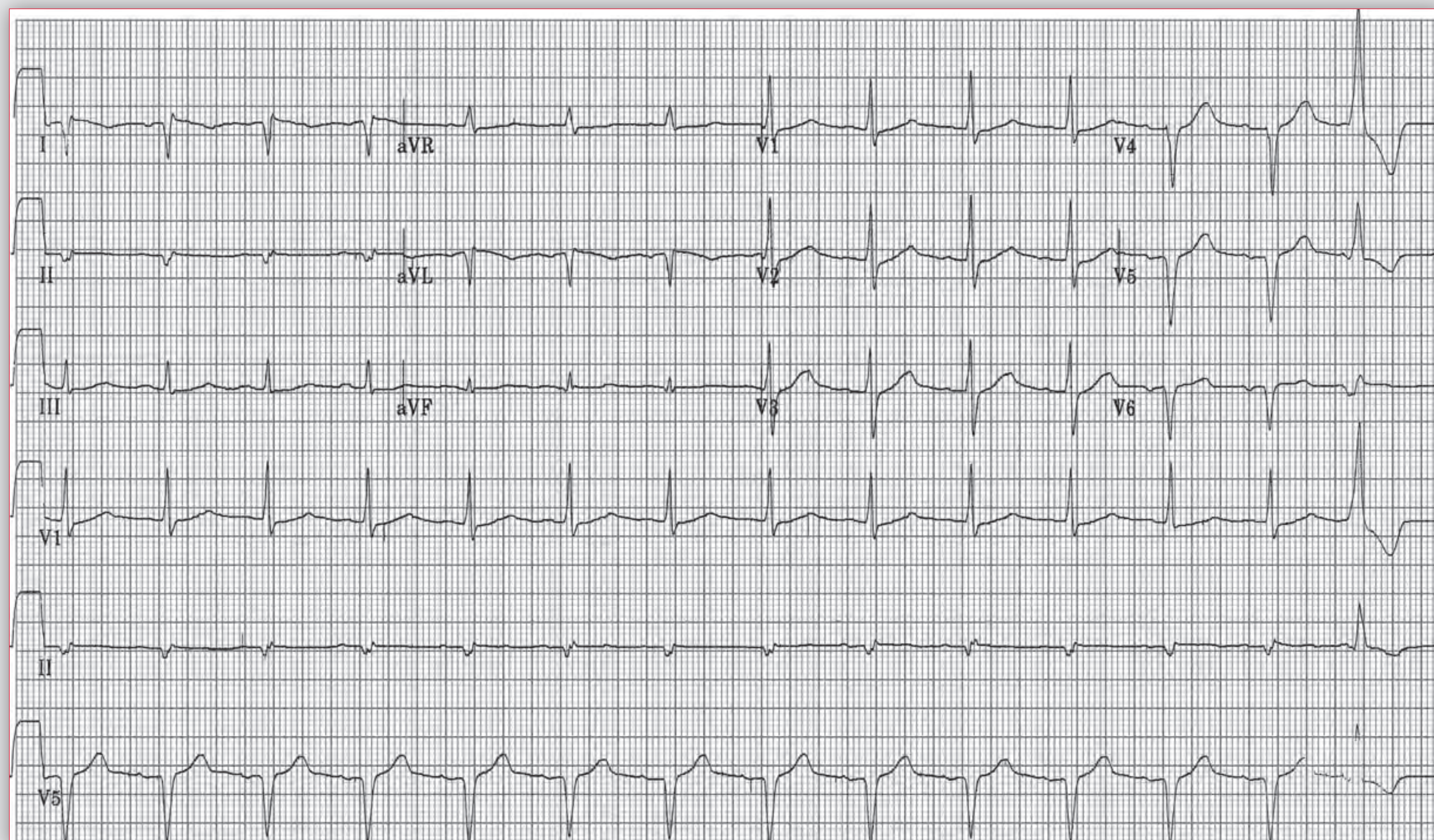
ECG 90 Analysis: Normal sinus rhythm, dextrocardia

There is a regular rhythm with a rate of 68 bpm. There is a P wave (+) before each QRS complex in leads I and III, where it appears to be negative. There is a stable PR interval (0.14 sec). P waves are not seen in the other leads. The QRS duration is normal (0.08 sec). The QT/QTc intervals are normal (380/405 msec). The axis is rightward between +90 and +180 (positive QRS complex in lead I and positive in lead aVF). In addition, there is reverse R-wave progression in leads V1–V6, *ie*, the R-wave amplitude progressively decreases (↓). One potential explanation for this pattern is right left arm lead switch as well as misplacement of leads V1–V6. However, this ECG is characteristic of dextrocardia, assuming it is certain that the leads were placed in the right position. The presence of dextrocardia could be confirmed on chest x-ray and also by placing the chest leads on the right side of the chest (and hence over the heart). ■

Core Case 91

A 15-year-old boy is referred to a pediatric cardiologist because his new pediatrician notes that his ECG (ECG 91A) is very abnormal. There is no history of any cardiac problems or symptoms. The cardiologist repeats the ECG and finds the same abnormalities. He obtains a chest x-ray, after which he repeats the ECG (ECG 91B) after making some changes.

ECG 91A

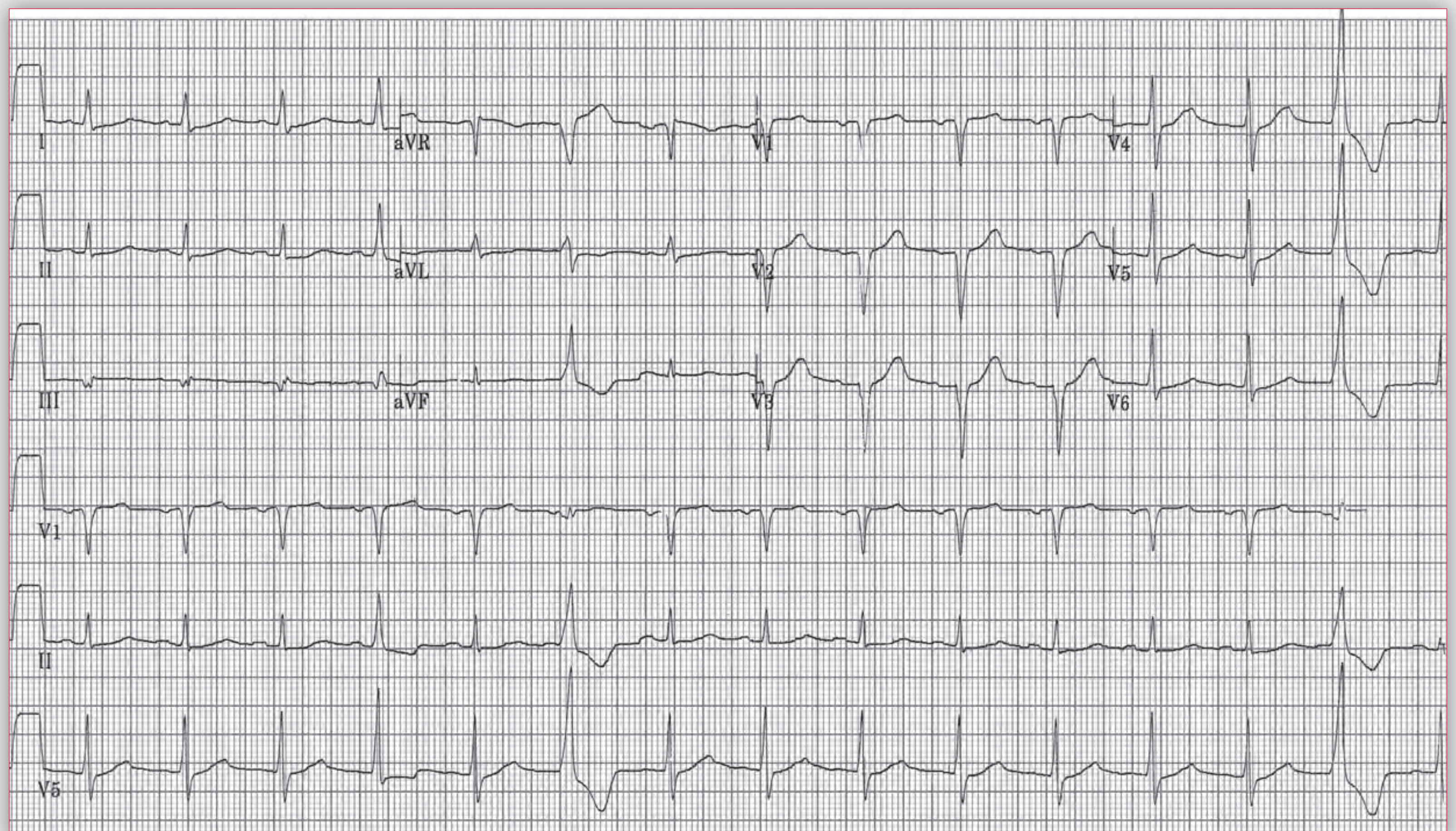


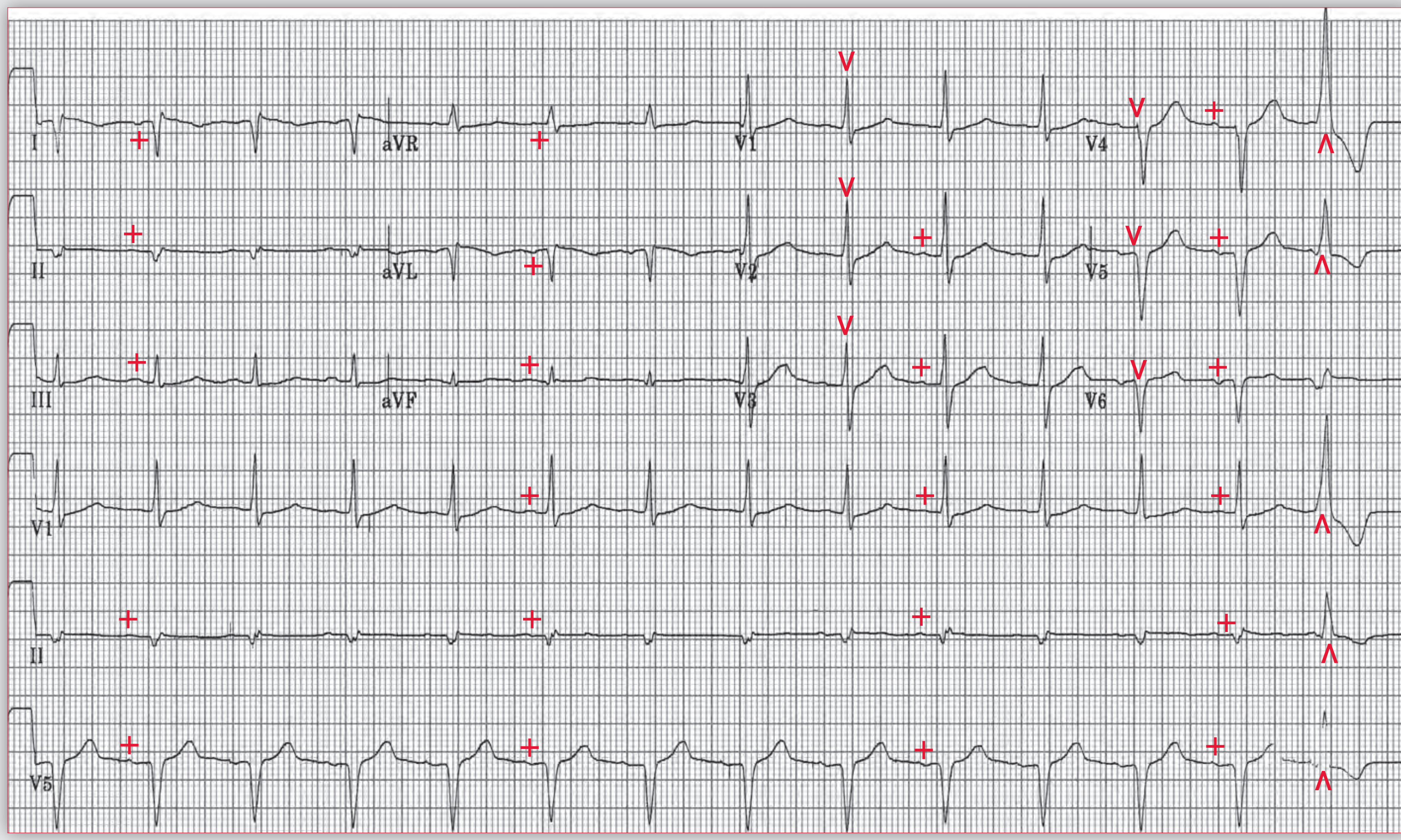
What is the abnormality seen?

What is the underlying cardiac problem?

What changes were made in when ECG 91B was recorded?

ECG 91B





ECG 91A Analysis: Normal sinus rhythm, dextrocardia (appears to be R-L arm lead switch, reversal of leads V1–V6)

There is a regular rhythm at a rate of 86 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). However, the P-wave morphology is abnormal, as there is a negative P wave in lead I as well as leads V5–V6 and a positive P wave in lead aVR. This is suggestive of an atrial rhythm. The QRS complex duration is normal (0.08 sec), but there is a rightward axis between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). In addition, there is a tall R wave in lead aVR. Together with the negative P wave in lead I, this pattern is suggestive of a right-left arm lead switch. Another cause for this pattern is dextrocardia. The QT/QTc intervals are normal (360/440 msec). The last QRS complex (^) is premature and has a wide and abnormal morphology. This is a premature ventricular complex.

The QRS complexes in leads V1–V6 are also abnormal, as there is reverse R-wave progression (v), *ie*, the R wave is tallest in lead V6 and becomes progressively shorter. The R wave is very small in lead V6. Indeed, the QRS complex morphology in lead V6 is what V1 should look like, and the morphology in lead V1 is what V6 should look like. Hence this is consistent with all the precordial leads reversed. However, it would be rather unusual for both the limb and chest leads to be placed in the wrong location. This ECG (91A) is strongly suggestive of dextrocardia.

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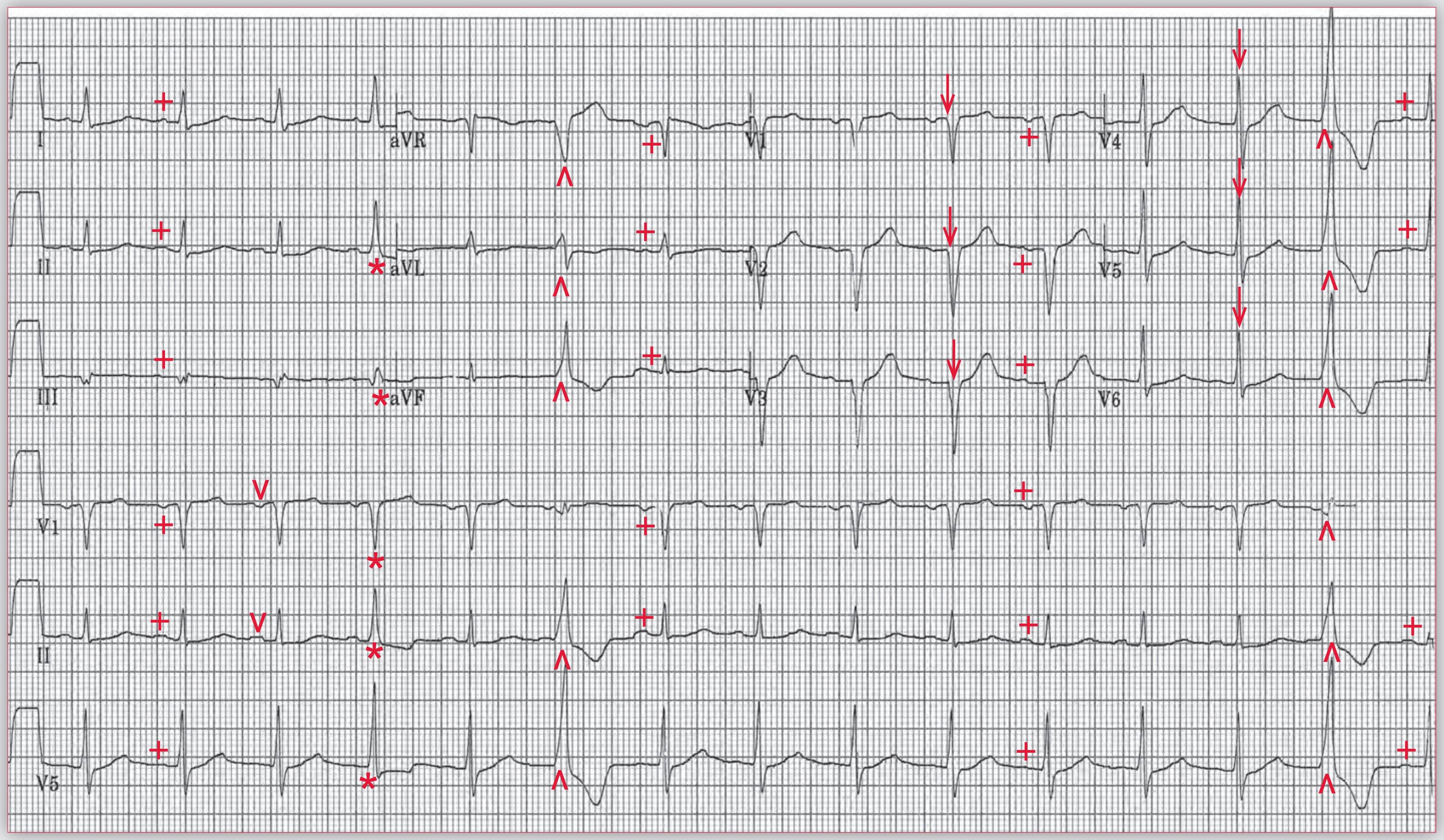


ECG 91B Analysis: Normal sinus rhythm, R-L arm lead switch, right sided leads in dextrocardia, premature ventricular complexes

The presence of dextrocardia is confirmed by **ECG 91B** that was actually recorded with the R and L arm leads switched and the chest leads placed on the right side of the chest. It can be seen that the ECG has P waves (+) that are normal, and there is normal R-wave progression (↓) across the precordium, with the R wave becoming progressively taller. The QRS complex duration is normal (0.08) and there is now a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are the same as ECG 91A.

The fourth QRS complex (*) is slightly premature and is slightly wider (0.10 sec) than the sinus complexes with a morphology that is slightly different from the sinus complexes. In addition, it is associated with an on-time P wave (v) with a shorter PR interval (0.14 sec). This is a fusion complex, *ie*, it is a premature ventricular complex that fused with the sinus node impulse coming through the normal AV node His-Purkinje system to activate the ventricle. The sixth and fourteenth QRS complexes (^) are also premature and there are no P waves before them. The fusion complex resembles both the premature ventricular complex as well as the sinus complex. They have a wide and usual morphology. These are premature ventricular complexes. ■

Notes

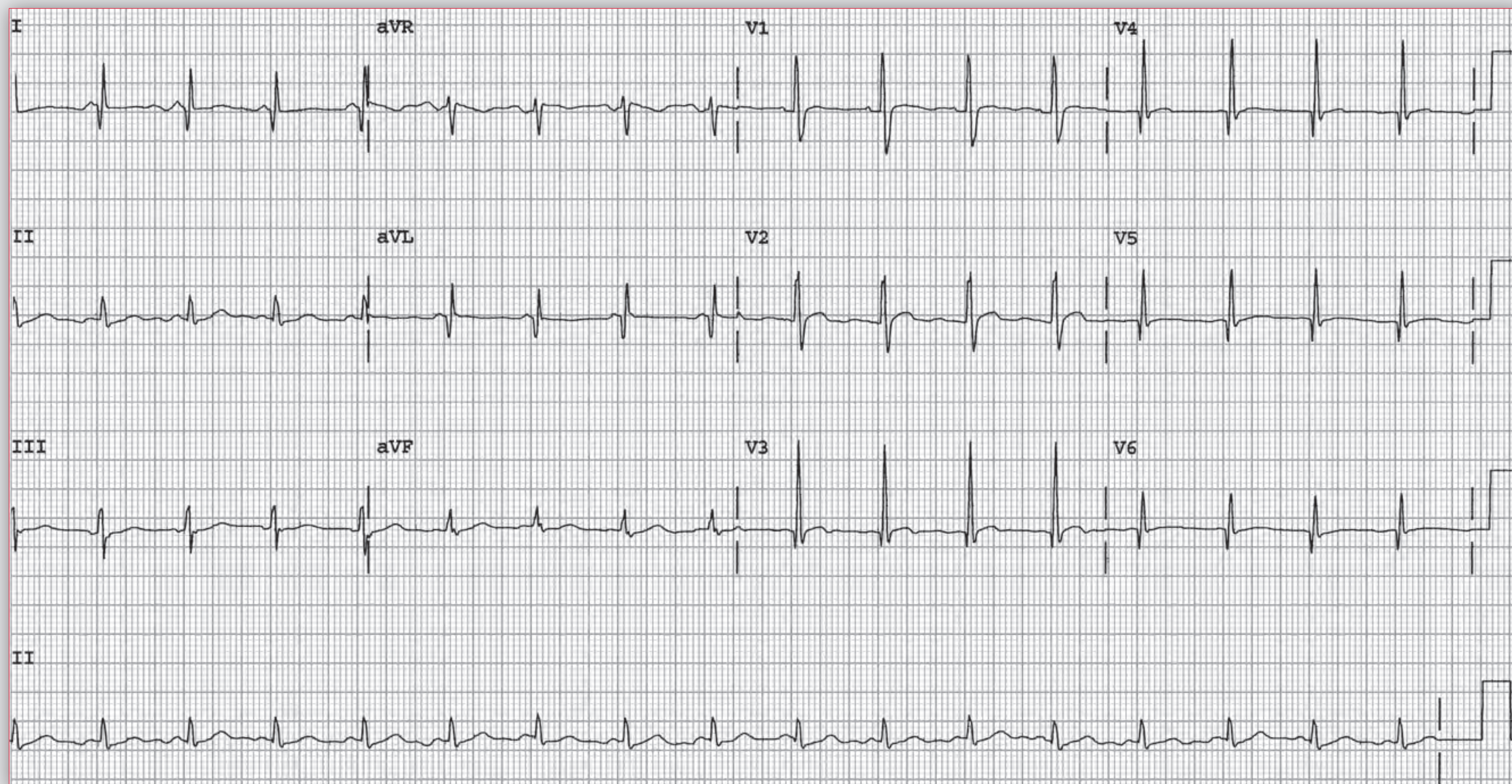
Core Case 92

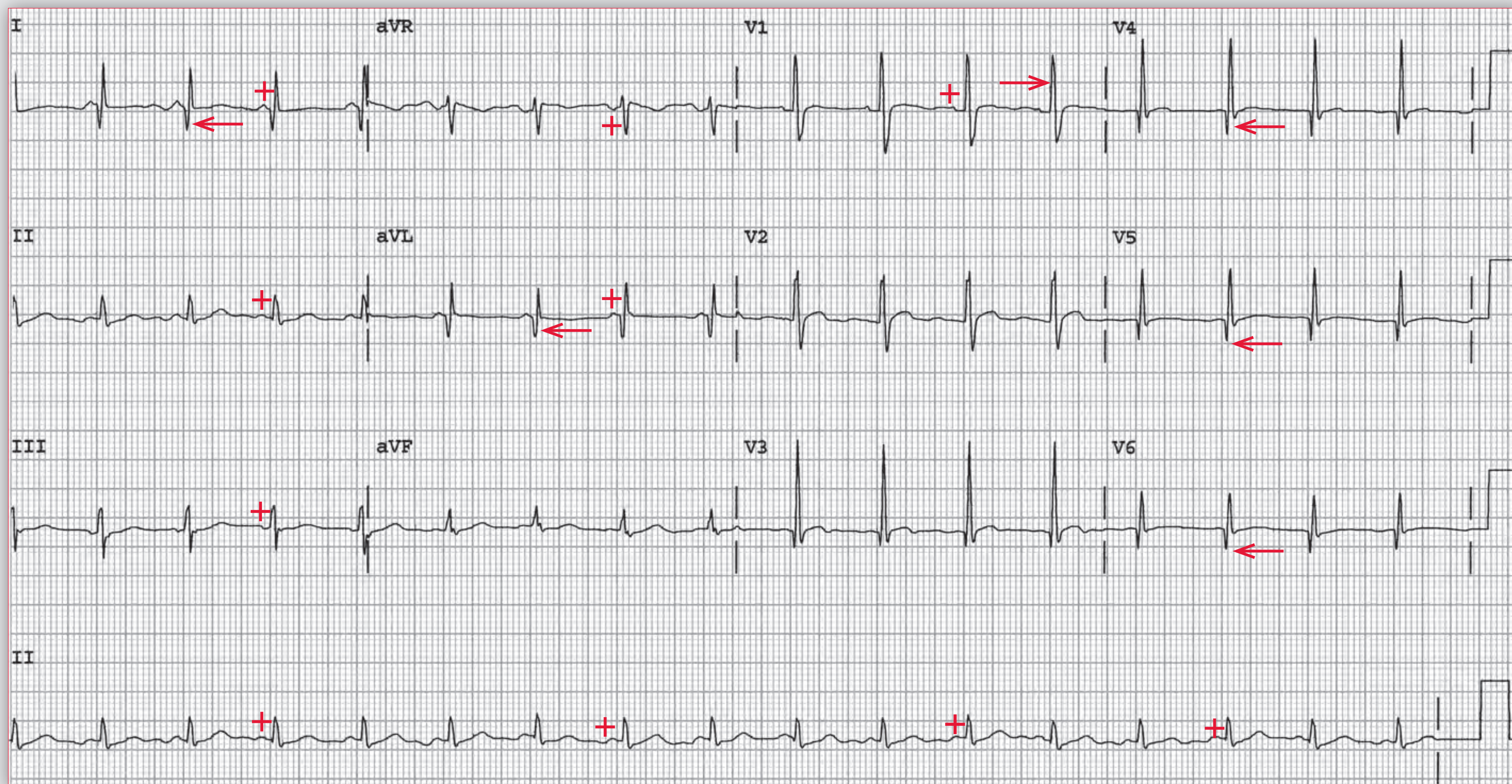
A 24-year-old male presents with palpitations. He has a known muscular dystrophy but is without known cardiac disease. His cardiac exam is otherwise normal. An ECG is obtained.

What abnormalities are noted on the ECG?

What type of muscular dystrophy does he have?

What other cardiac diagnoses may result in similar ECG abnormalities?





ECG 92 Analysis: Sinus tachycardia, short PR interval, posterolateral myocardial infarct pattern, Duchenne's muscular dystrophy

There is a regular rhythm with a rate of 110 bpm. There is P wave (+) before each QRS complex with a stable PR interval (0.12 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is sinus tachycardia, which may be the result of enhanced AV nodal conduction or a preexcitation syndrome termed Lown-Ganong-Levine syndrome.

The QRS complex duration is normal (0.08 sec) and the axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (320/430 msec). There are Q waves in leads I, aVL, and V4–V6 (←) as well as a tall R wave in lead V1 (→). There are a number of etiologies for a tall R wave in lead I, including:

1. Posterior wall MI (usually associated with inferior MI)
2. Wolff-Parkinson-White pattern (short PR and widened QRS with delta wave)
3. Hypertrophic cardiomyopathy (septal hypertrophy with prominent septal Q-waves and T-wave abnormalities)
4. Early transition (counterclockwise rotation)
5. Duchenne's muscular dystrophy (posterolateral infarct pattern)
6. Dextrocardia (reverse R-wave progression V1–V6, right axis, negative P and T waves in leads I and aVL, and positive in lead aVR)
7. Lead switch (leads V1, V2, V3)
8. Right-sided leads
9. Normal variant due to counterclockwise rotation (early transition)

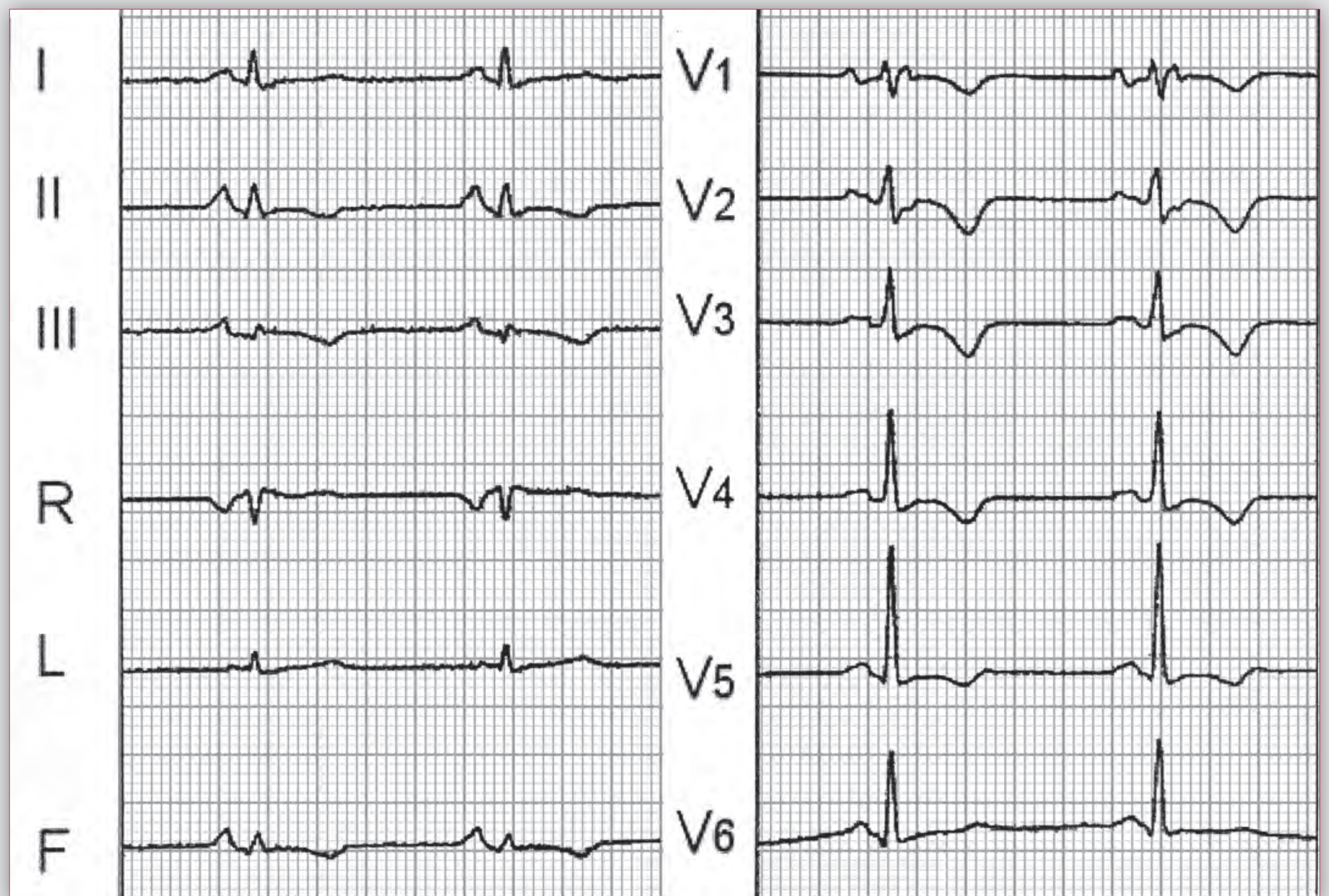
This ECG has a pattern of a posterolateral infarction. Thus this is an ECG that is characteristic of Duchenne's muscular dystrophy, as the cardiac fibrosis develops first in the posterolateral part of the heart and then progresses to involve the entire myocardium, producing a dilated cardiomyopathy. ■

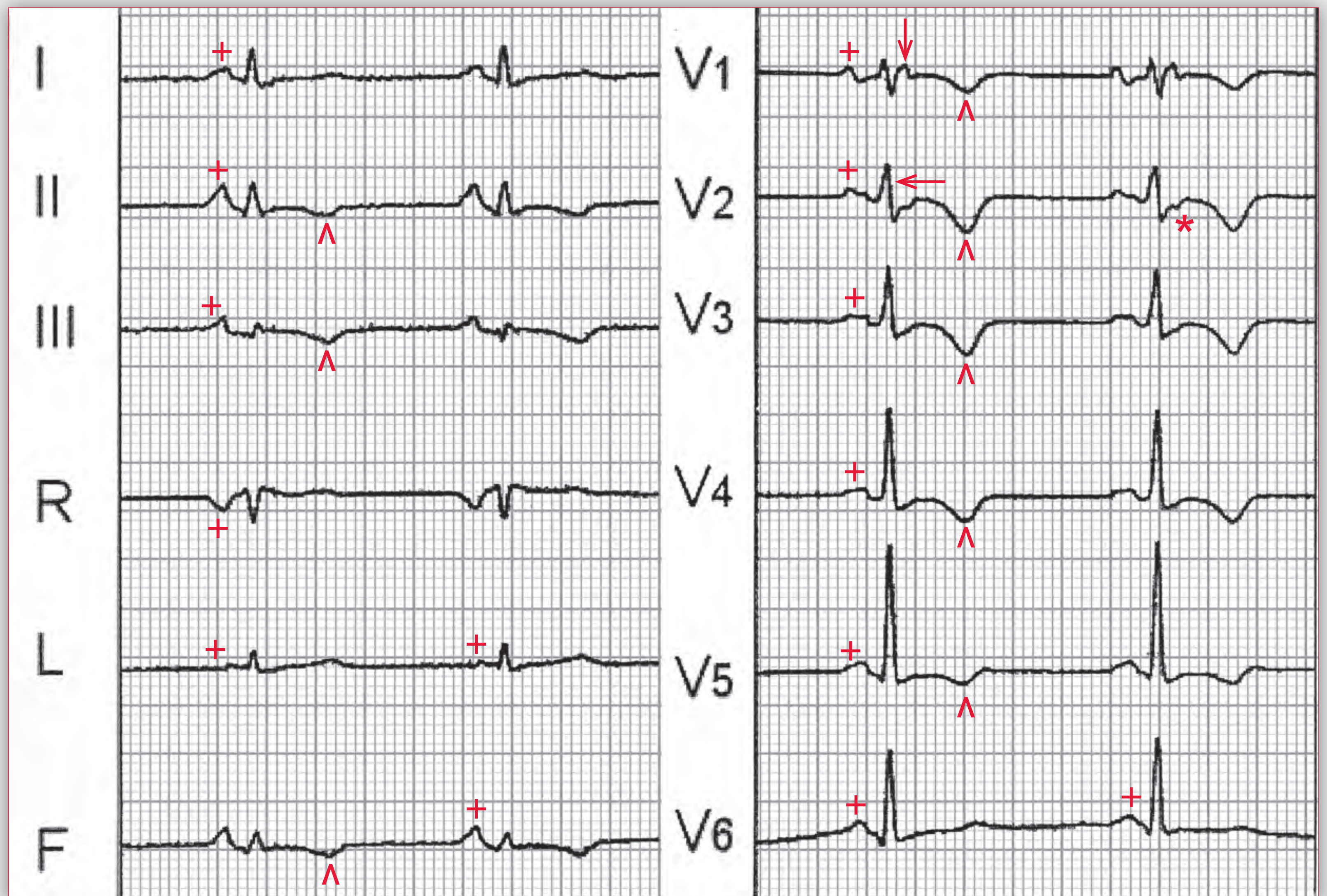
Notes

Core Case 93

A 24-year-old professional football player is undergoing a routine screening exam. His medical history is unremarkable. It is noted that his family is originally from northern Italy. He does remember being told about some relatives having experienced sudden death. However, he is not aware of any details about this. On review of symptoms, however, he reports occasional palpitations, without associated symptoms, for the past 2 months. Based on this, an ECG is obtained.

What abnormality is evidenced by the ECG, and what diagnosis is suggested?





ECG 93 Analysis: Sinus bradycardia, early transition, nonspecific T-wave abnormalities. Epsilon wave in leads V1–V2, arrhythmogenic right ventricular cardiomyopathy (dysplasia)

There is a regular rhythm at a rate of 54 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6, and hence this is a sinus bradycardia. The QRS complex duration is normal (0.08 sec) and the axis is normal between 0° and +90° (positive QRS complex in lead I and aVF). There is a tall R wave in lead V2 (←), consistent with early transition or counterclockwise rotation. This is established by imagining the heart as being viewed from under the diaphragm. When there is counterclockwise rotation in the horizontal plane, the left ventricular forces are shifted anteriorly and occur earlier in the precordial leads, presenting with a tall R wave in lead V2. There are nonspecific T-wave inversions (^) noted in leads II, III, and aVF as well as leads V1–V5. The QT/QTc intervals are normal (440/425 msec). The QRS complex has a normal morphology except in lead V1. Noted at the end of the QRS complex is what looks like an R′ (↓), although it has a rounded morphology. The QRS complex duration in this one lead appears to be prolonged (0.12 sec) as a result of this waveform. This waveform can also be seen in lead V2 (*), and there it appears to be superimposed on the terminal portion of the QRS complex. Although this looks like a right bundle branch block (RBBB), the QRS duration in all the other leads is normal and there are no broad S waves in leads I and V5–V6, which would be typical of a RBBB. This abnormality is termed an epsilon wave and it is characteristic of arrhythmogenic right ventricular dysplasia (ARVD) or cardiomyopathy (ARVC). This is a genetic cardiomyopathy classically affecting the right ventricle (RV), identified histologically by a mononuclear infiltrate, degenerating myocytes, and fibrofatty replacement of myocardial tissue. The epsilon wave results from this RV abnormality and is best seen in the precordial leads over the RV, *ie*, V1–V2.

In most cases, ARVC is clinically silent in early stages of disease. Many patients are asymptomatic, and ARVC is suspected because of the presence of nonspecific electrocardiographic or echocardiographic abnormalities or ventricular arrhythmias on Holter or exercise testing in the context of a positive family history of ARVC or sudden cardiac death. The first presenting symptom may be sudden cardiac arrest. ARVC may also present with palpitations or syncope that are due to ventricular arrhythmias. Other symptoms include dyspnea or atypical chest pain. Infrequently, the patient may present with right-sided heart failure. Symptoms often first occur at a mean age of 30 (range 10 to 50 years old). Thirty percent of cases are familial, more often autosomal dominant. An autosomal recessive form of the disease, attributed to mutations in plakoglobin or desmoplakin (genes encoding proteins comprising desmosomes) has also been described. It is over-represented in sudden cardiac death among athletes, particularly in northern Italy. Some degree of left ventricular (LV) involvement is seen in most cases, though a subset of cases are characterized with early and more severe left-sided involvement as compared with the RV.

continues

Major criteria for the diagnosis of ARVC include:

1. Severe dilatation and reduction of RV ejection fraction with no (or only mild) LV impairment
2. Localized RV aneurysms (akinetic or dyskinetic areas with diastolic bulging)
3. Severe segmental dilatation of the RV
4. Fibrofatty replacement of myocardium on endomyocardial biopsy (or seen on an MRI with contrast)
5. Epsilon waves or localized prolongation (> 110 ms) of the QRS complex in right precordial leads (V1–V3)
6. Familial disease confirmed at necropsy or surgery

Minor criteria include:

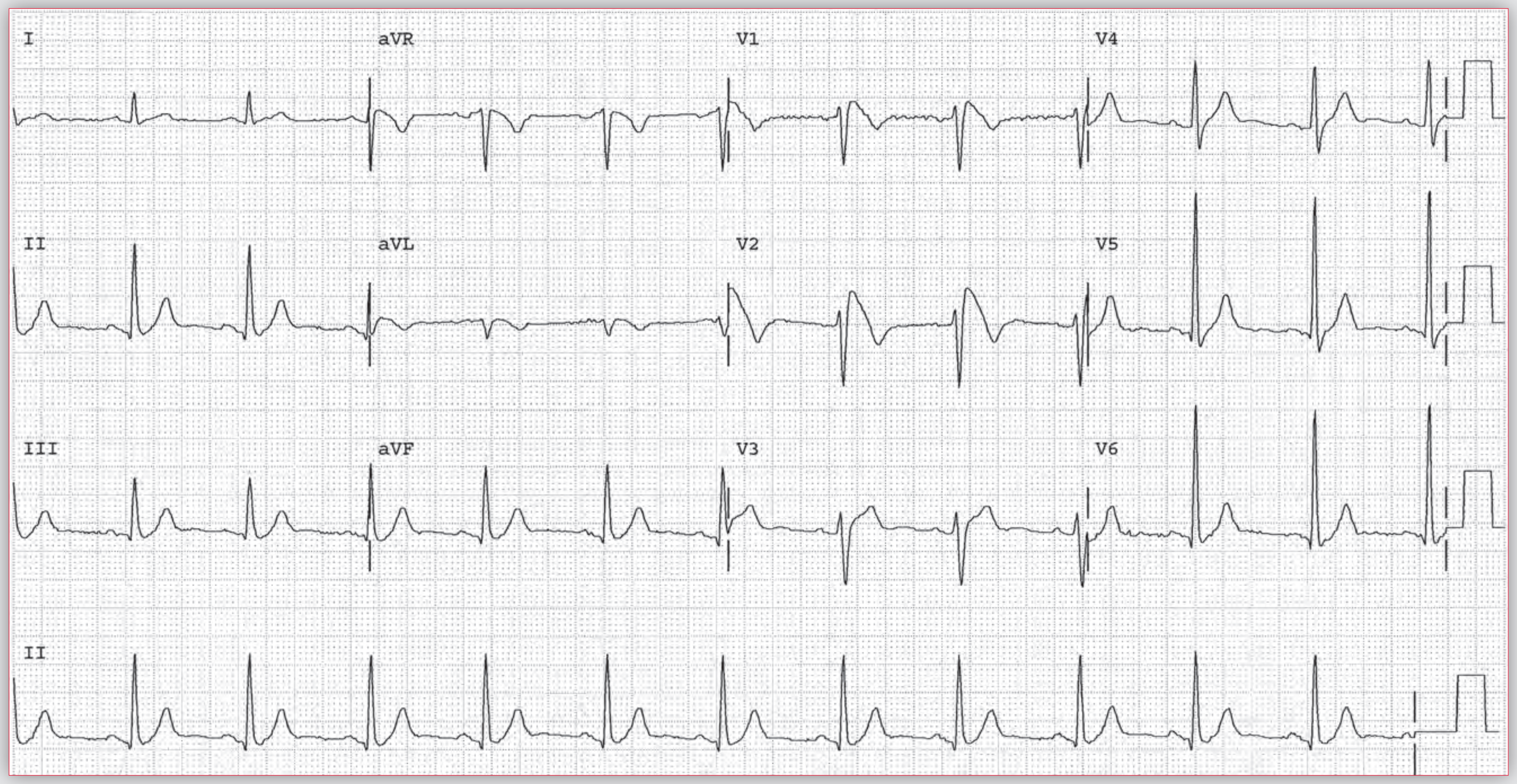
1. Mild global RV dilatation and/or ejection fraction reduction with normal LV
2. Mild segmental dilatation of the RV
3. Regional RV hypokinesia
4. Inverted T waves in right precordial leads (V2 and V3) (people aged > 12 years, in absence of RBBB)
5. Late potentials (SAECG)
6. LBBB-type ventricular tachycardia (sustained and nonsustained) (ECG, Holter, exercise)
7. Frequent ventricular extrasystoles (> 1000 per 24 hours) (Holter)
8. Family history of premature sudden death (< 35 years of age) due to suspected ARVC
9. Familial history (clinical diagnosis based on present criteria)

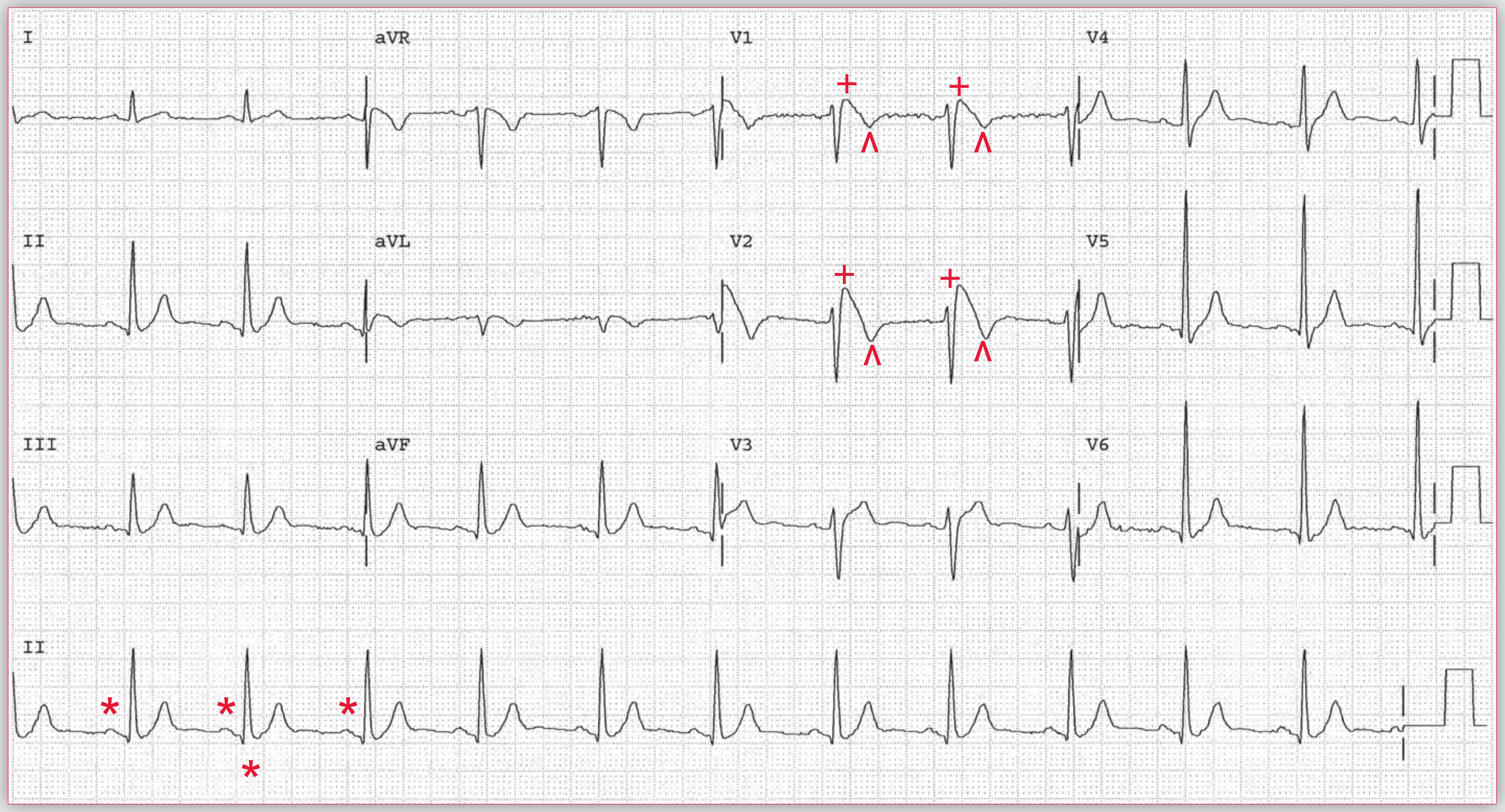
A definite diagnosis is established if there are 2 major criteria present or 1 major and 2 minor criteria or 4 minor criteria. A possible diagnosis is present if there is 1 major and 1 minor criteria present or 3 minor criteria from different categories.

A 30-year-old male suddenly loses consciousness while at dinner. He spontaneously regains consciousness within several seconds. He is taken to a local hospital.

Initial evaluation reveals an awake and alert male with normal vital signs. An ECG is obtained as part of his workup.

What abnormality is noted, and what diagnosis is suggested to explain his syncopal event?





ECG 94 Analysis: Normal sinus rhythm, normal axis, type 1 Brugada pattern (pseudo right bundle branch block)

ECG 94: There is a regular rhythm at a rate of 72 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec). The axis in the frontal plane is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/395 msec). The QRS morphology is normal except in leads V1–V2. These leads show an elevated J point and ST segment (≥ 2 mm) (+) that descends slowly with an upward convexity to an inverted T wave (^). This has been termed a pseudo right bundle branch block (RBBB) pattern and is typical of a classic type 1 Brugada pattern.

The mechanism for the Brugada pattern is a genetic abnormality of the SCN5A sodium channel gene that affects sodium influx during the early part of phase 2 of the action potential. This alters the action potential of some, but not all of the ventricular myocardial cells. The ventricular myocardium is composed of at least three electrophysiologically distinct cell types: epicardial, endocardial, and M cells. The ST-segment elevation and T-wave inversions seen in the right precordial leads in Brugada pattern are thought to be due to an alteration in the action potential in the epicardial and possibly the M cells, but not the endocardial cells. The resulting dispersion of repolarization across the ventricular wall, which is most pronounced in the right ventricle, results in a transmural voltage gradient that is manifested in the ECG as J-point and ST-segment elevation.

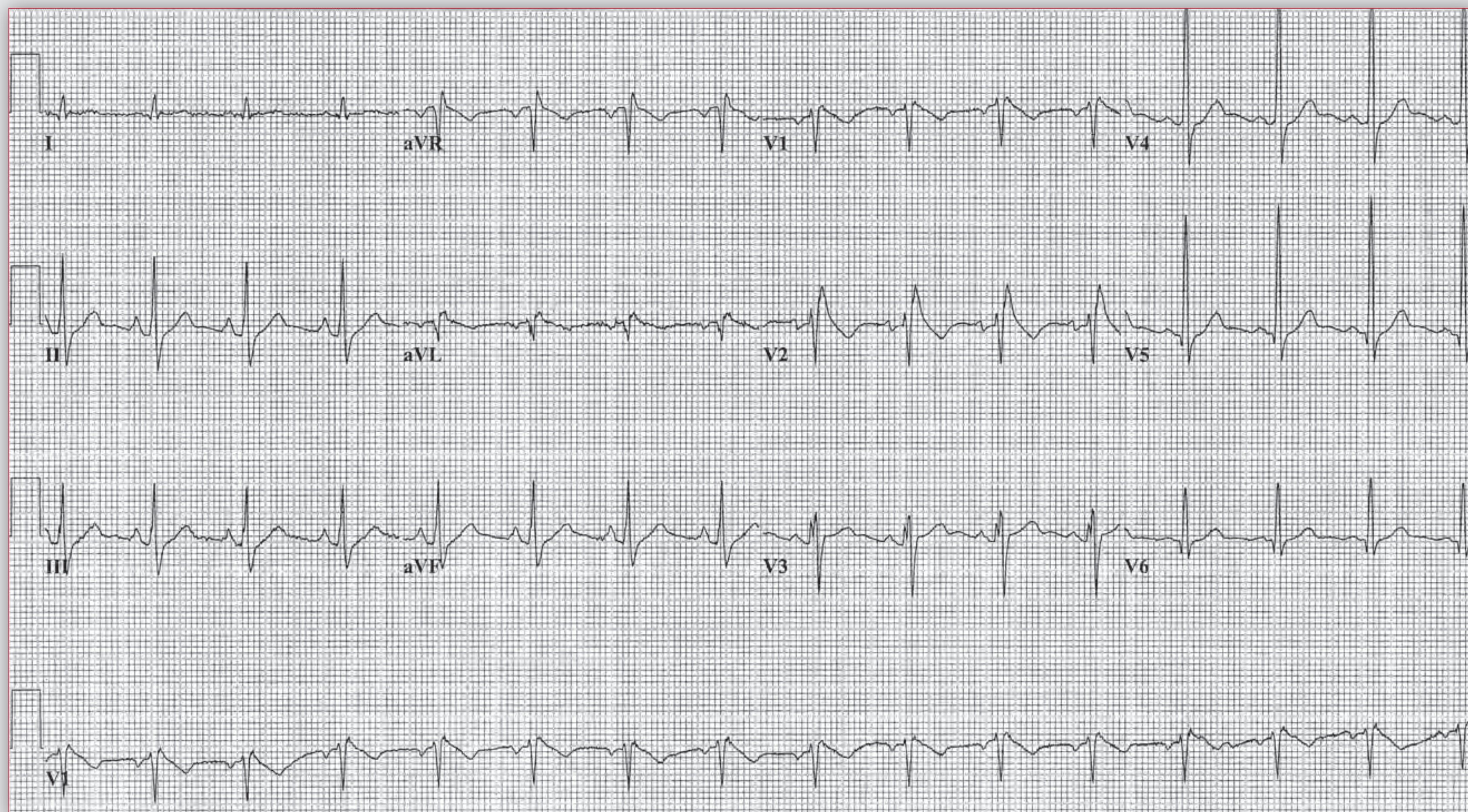
Patients with arrhythmia associated with the Brugada pattern have the Brugada syndrome. All clinical manifestations of the Brugada syndrome are related to life-threatening ventricular arrhythmias (often manifest as syncope or sudden cardiac arrest). Sudden cardiac arrest may be the first and only clinical event in the Brugada syndrome.

Although pharmacologic therapy, particularly quinidine, may be of some benefit, the therapy of choice for patients with symptoms is an implantable cardioverter-defibrillator (ICD). ■

Core Case 95

An 18-year-old man is referred to a cardiologist by his primary doctor. The primary physician uncovered a family history of multiple unexplained deaths and in response

ECG 95A

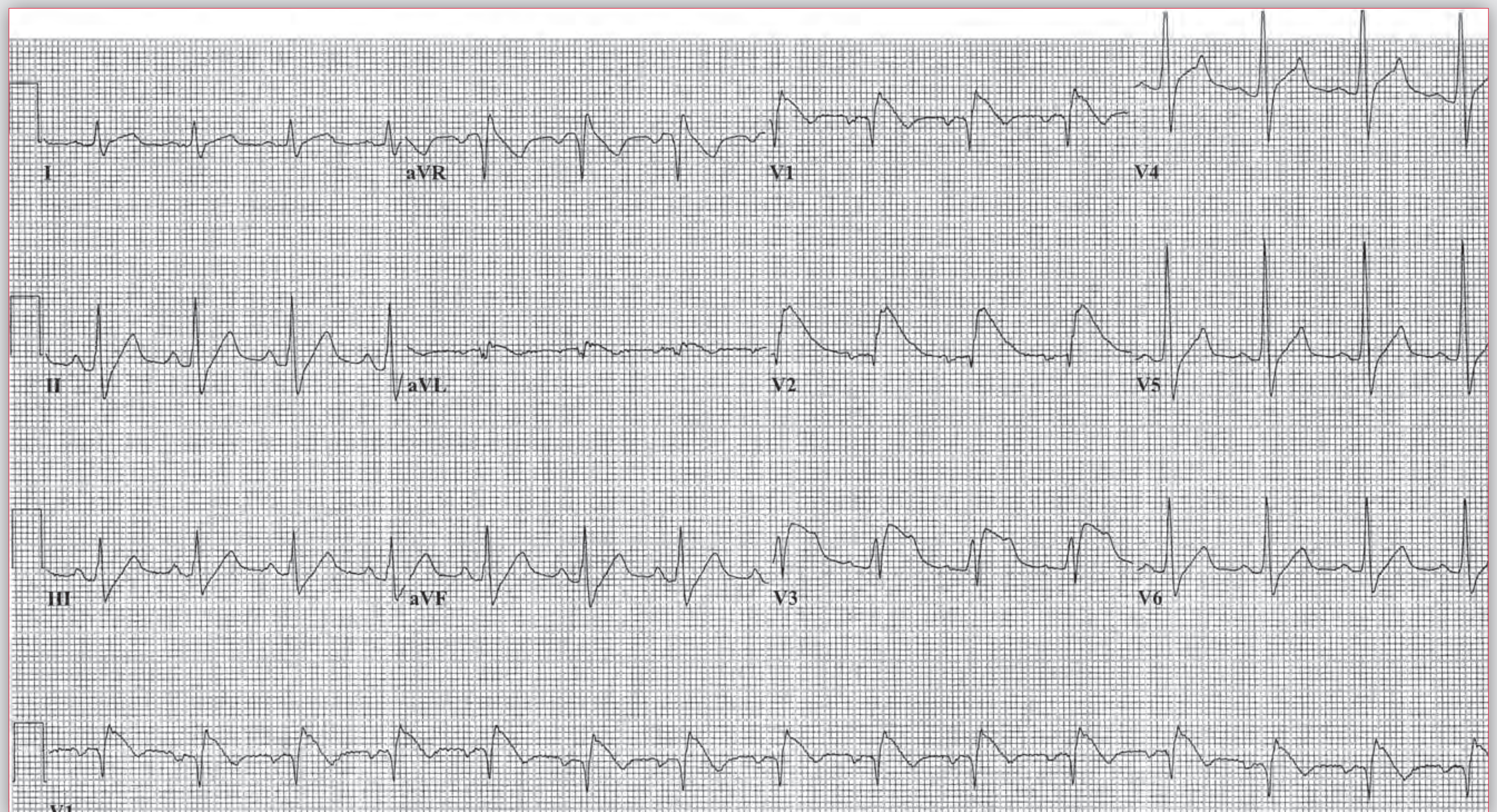


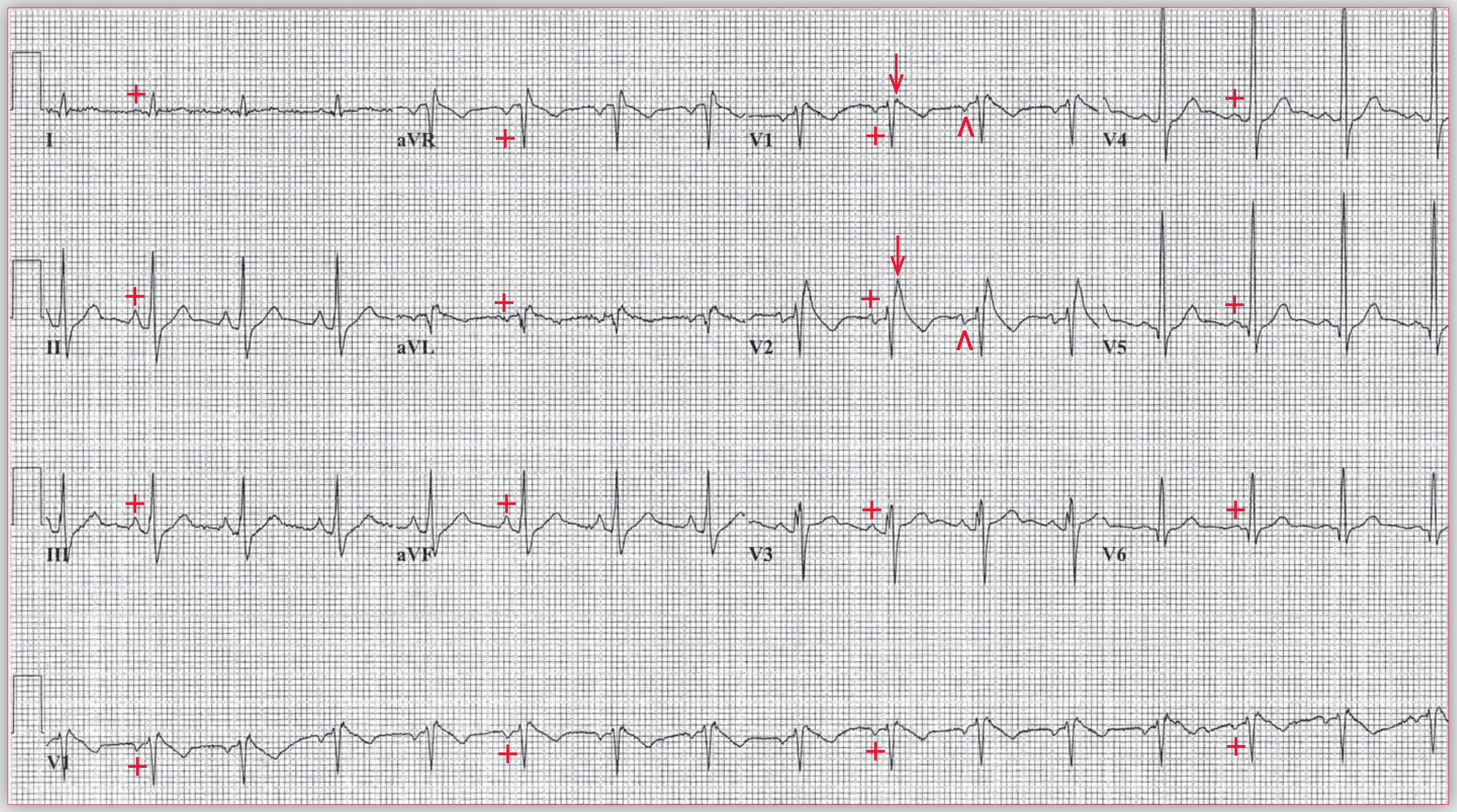
obtained an ECG from the patient (ECG 95A).

Upon review of the history and ECG, the patient undergoes a diagnostic test, during which the second tracing (ECG 95B) is obtained.

What abnormality is noted in the tracings, what diagnostic test was likely performed, and what is the patient's diagnosis?

ECG 95B





ECG 95A Analysis: Normal sinus rhythm, ST-segment elevation in leads V1–V2 (pseudo right bundle branch block pattern suggestive of a Brugada pattern)

ECG 95A shows there is a regular rhythm with a rate of 92 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec). The P waves are positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Therefore, this is a sinus rhythm. The P wave is negative in lead V1 and biphasic in lead V2 (^), suggesting left atrial hypertrophy or a left atrial abnormality.

The QRS duration is normal (0.08 sec). The axis in the frontal plane is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/400 msec). The QRS morphology is normal except in leads V1–V2. There is an RSR′ morphology in leads V1–V2 (↓), which is commonly termed an incomplete right bundle branch block (RBBB). However, the presence of a Brugada pattern should be suspected, especially if there is a patient or family history of syncope or ventricular tachyarrhythmias, as this is not a typical pattern for a RBBB as the downslope of the complex (R′) is slow or prolonged.

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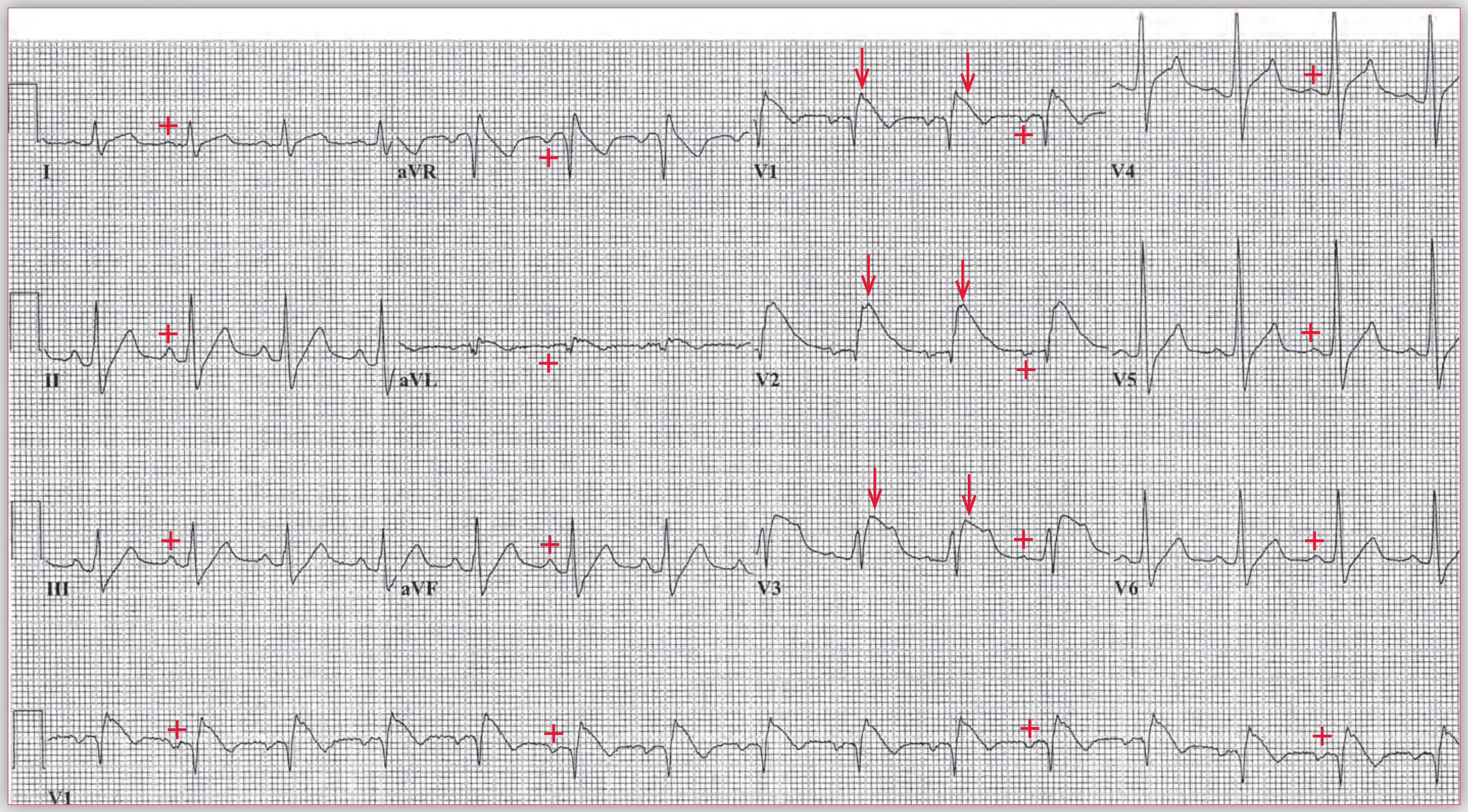


ECG 95B Analysis: Normal sinus rhythm, type 1 Brugada pattern

ECG 95B is from the same patient as ECG 95A. It was obtained after the infusion of intravenous procainamide. There is a P wave before each QRS complex, and the P waves are (+) positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration, axis and QT interval are the same as in ECG 95A. Noted are marked abnormalities in the J point and ST segment that are present in V1–V3 (↓). These leads show an elevated J point and ST segment (≥ 2 mm) that descends slowly with an upward convexity to an inverted T wave. This has been termed a pseudo RBBB pattern and is typical of a coved type 1 Brugada pattern.

Among patients with an abnormal ECG (abnormal V1–V2) suggesting a Brugada pattern, a Brugada type 1 ECG pattern can occasionally be unmasked by sodium-channel blockers. Pacing, vagal maneuvers, and increased α-adrenergic tone also may provoke the typical Brugada ECG changes. Other factors that can unmask or modulate the ECG pattern are β-blockers, tricyclic or tetracyclic antidepressants, lithium, local anesthetic agents, fever, hypokalemia, hyperkalemia, hypercalcemia, and alcohol and cocaine toxicity.

Reported guidelines for the use of an ICD in patients with a Brugada pattern include:

1. ICD implantation is recommended in all those with a Brugada pattern on the ECG who have had a prior cardiac arrest.
2. ICD implantation is recommended for those with a Brugada pattern who have a history of VT that did not result in cardiac arrest.
3. ICD implantation may be indicated for those with a Brugada pattern who have had a history of syncope. Other causes of syncope, such as typical vasovagal events, bradycardia, or neurologic causes, must be excluded before proceeding to ICD implantation. ■

Notes

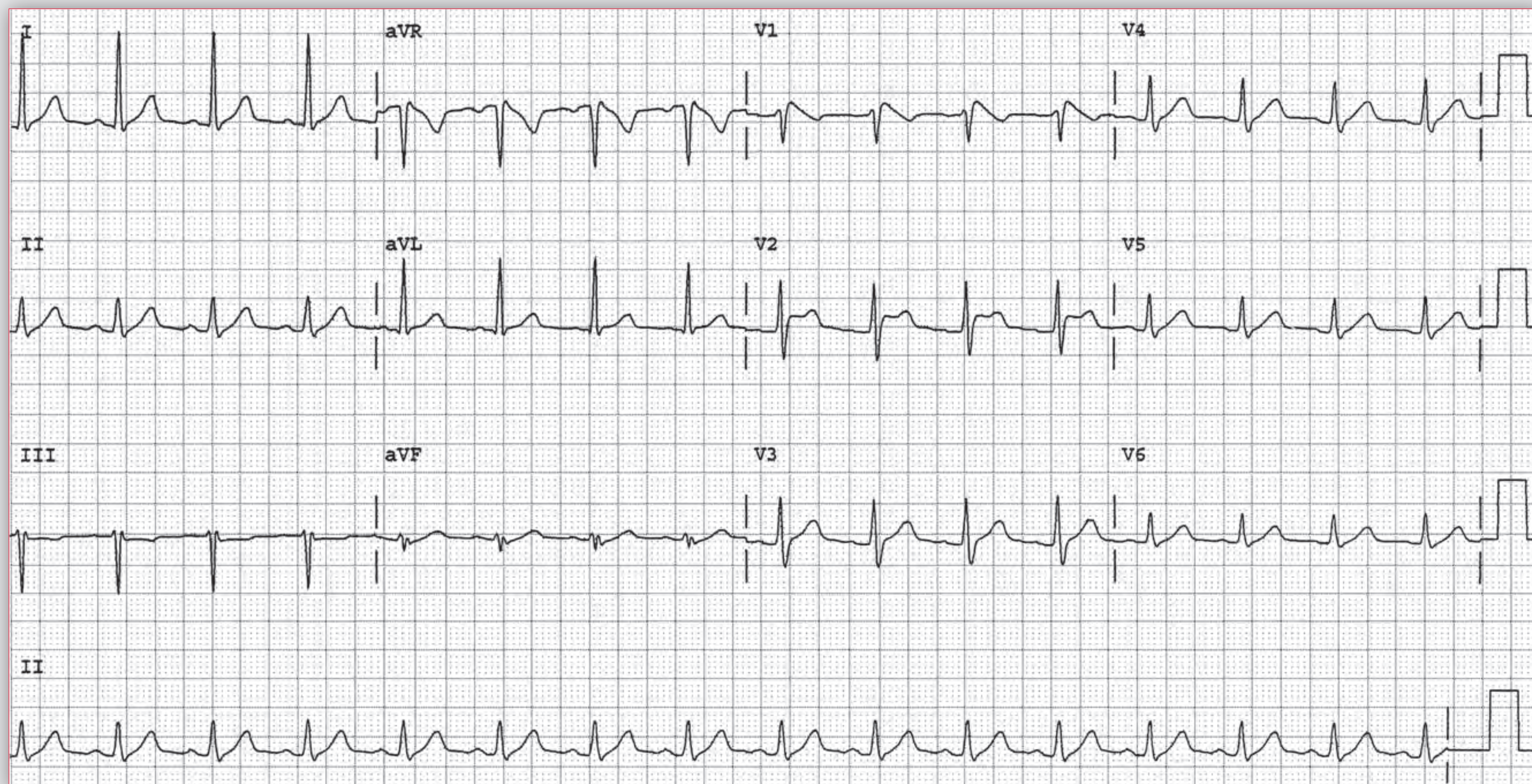
A 24-year-old man is undergoing a routine examination with a new primary care doctor. During the interview, he reveals that his father died suddenly of unexplained causes when he was very young. The patient himself denies ever having had palpitations or syncope. He is an active male without physical limitations.

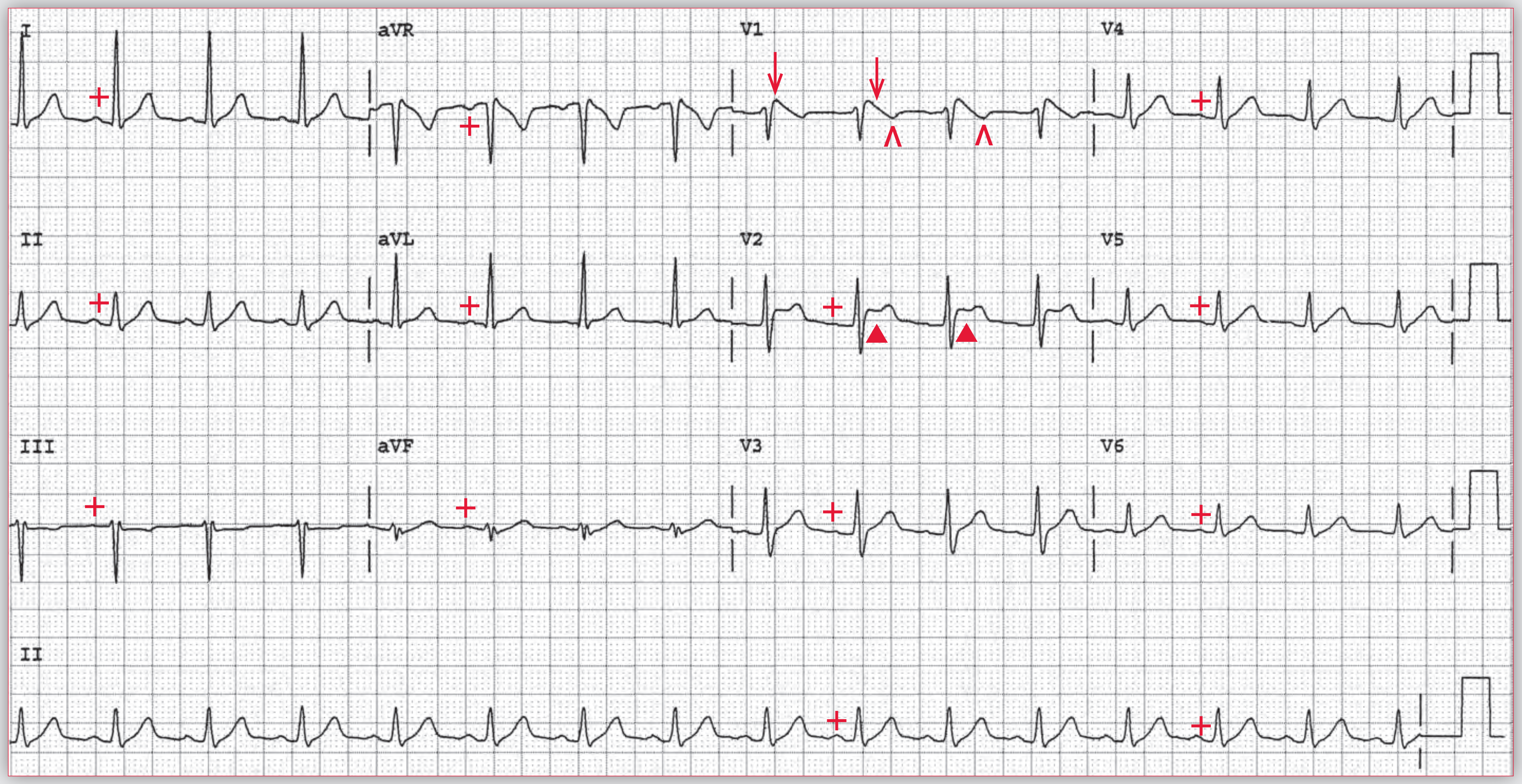
As part of his evaluation, an ECG is obtained.

What abnormalities are noted?

What do these suggest about the patient's family history and a possible diagnosis for the patient himself?

What further testing, if any, is indicated?





ECG 96 Analysis: Sinus tachycardia, type 2 Brugada pattern

There is a regular rhythm with a rate of 100 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4–V6 and negative in lead aVR. Therefore, this is a sinus tachycardia.

The QRS complex duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/440 msec). The QRS morphology is normal except in leads V1–V2. There is an RSR′ morphology in lead V1 (↓), suggesting a right bundle branch block (RBBB). However, the downslope of the complex is slow. It descends slowly with an upward convexity to an inverted T wave (^). This is not typical for a RBBB. In addition, there are no broad S waves in leads I or V5–V6. Not uncommonly this is termed an incomplete RBBB or an atypical RBBB.

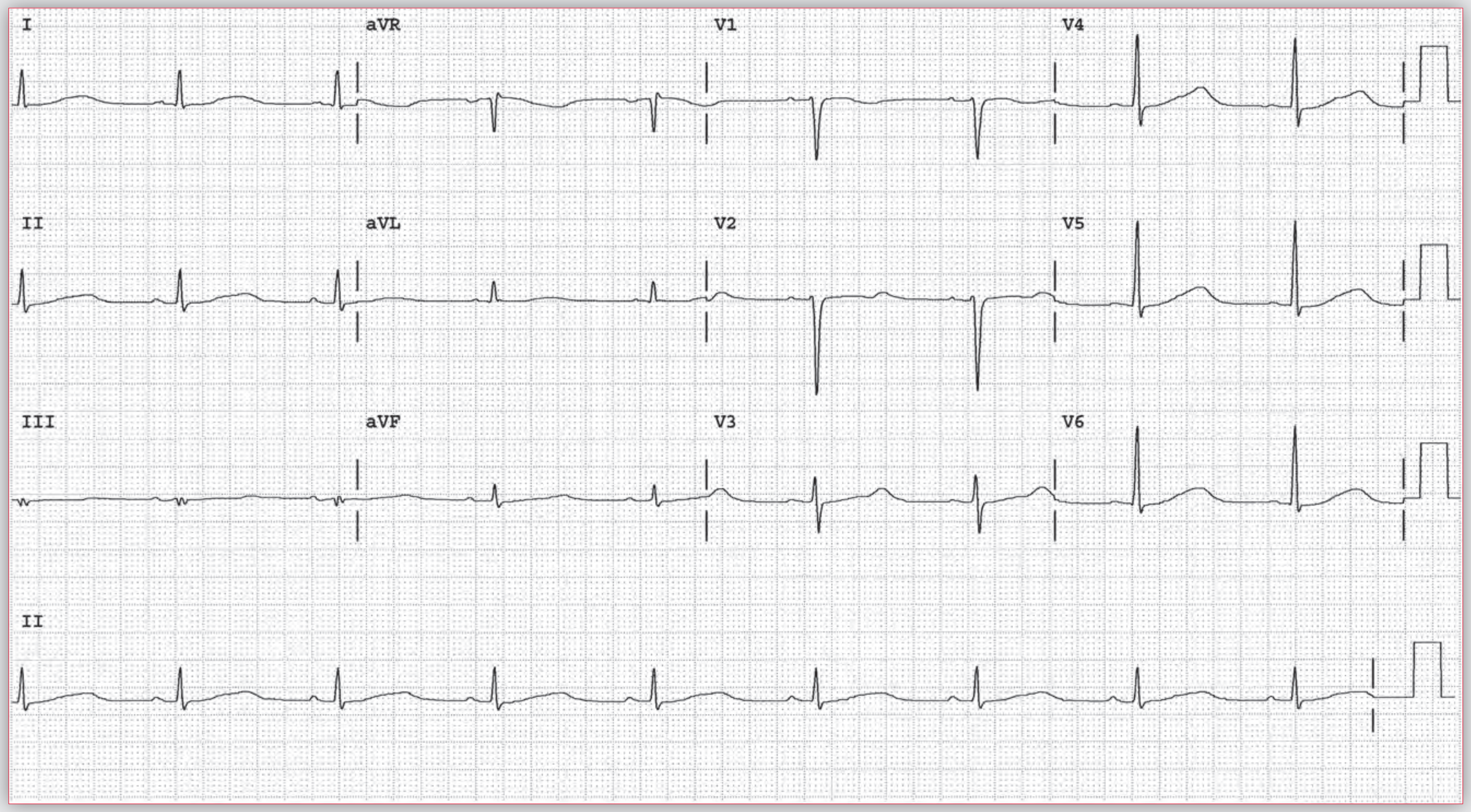
There is also an abnormal ST segment in lead V2 with J-point and ST-segment elevation (▲). It has a morphology that has been termed saddle-backed. The saddle-back ST-T wave configuration, in which the elevated ST segment descends toward the baseline, then rises again to an upright or biphasic T wave, is characteristic of type 2 and 3 Brugada patterns. In type 2, the ST-segment elevation ≥ 1 mm, and it is < 1 mm in type 3. Hence this is a type 2 Brugada pattern. In type 2 and 3 Brugada patterns, the diagnosis of Brugada syndrome is supported by documenting the conversion of the type 2 or 3 pattern to the type 1 pattern upon challenge with a sodium-channel blocker. The diagnosis is made when this finding is combined with documentation of ventricular arrhythmia, unexplained syncope, nocturnal agonal respiration, or family history of sudden death or a type 1 Brugada pattern. ■

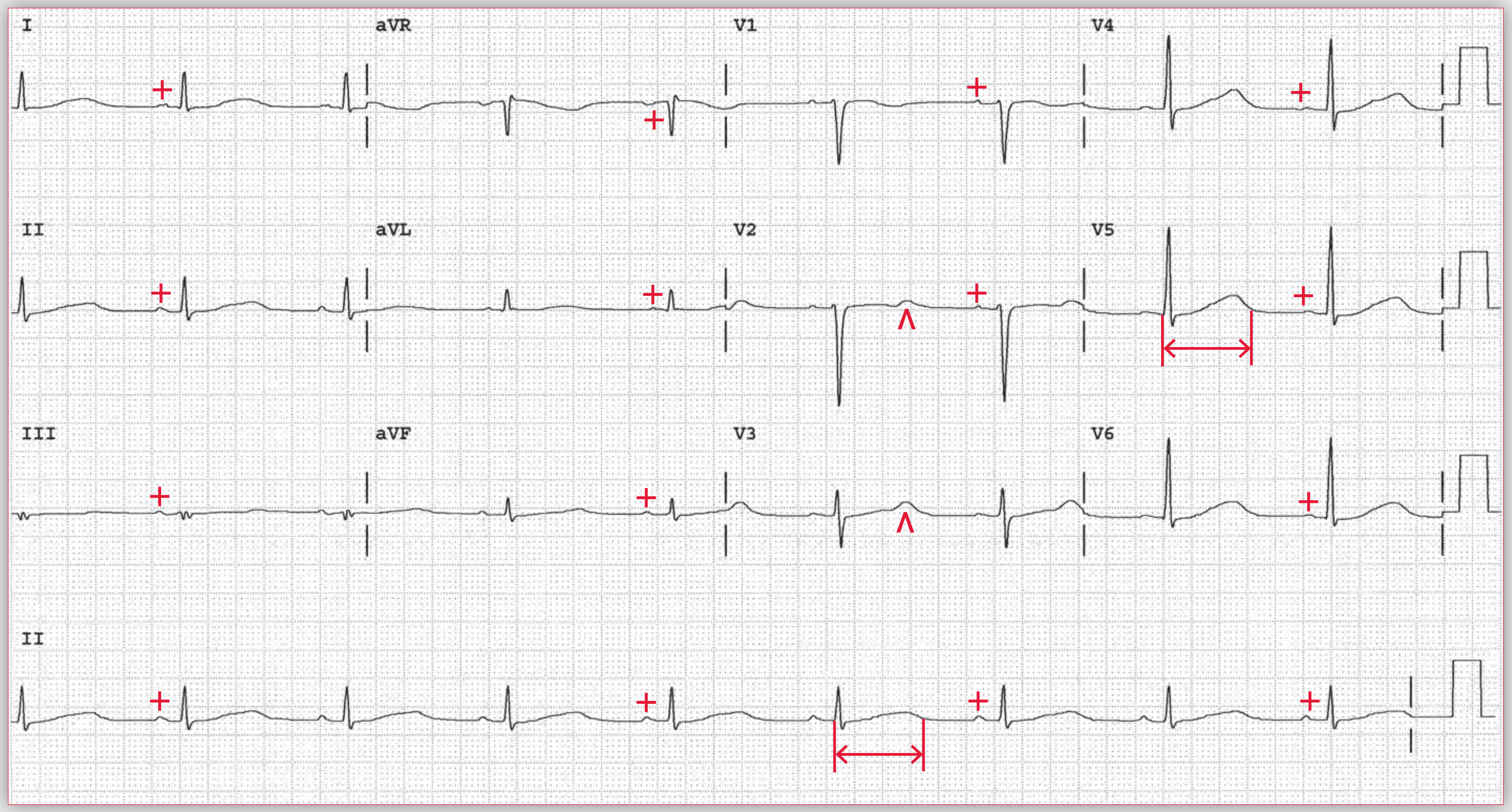
Notes

A 28-year-old male presents to his primary doctor with intermittent palpitations. He is a competitive athlete and noted a relation of symptoms to workouts. An ECG is obtained and read as normal.

One week later, he suffers an episode of syncope resulting in a motor vehicle accident. On review of his prior medical history, the outpatient ECG is re-interpreted.

What abnormality on the outpatient ECG suggests the cause of his symptoms and ultimate syncope?





ECG 97 Analysis: Sinus bradycardia, prolonged QT interval, QT-U wave in leads V2–V3

There is a regular rhythm at a rate of 52 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). Noted is a long QT interval (↔) (620 msec) and the rate-corrected QT (QTc) is 580 msec. As seen in leads V4–V6, there is a very broad T wave. Hence the QT prolongation is the result of prolonged depolarization, and this is a long QT syndrome (LQTS). Also noted in leads V2 and V3 is a prominent U wave (^) that is on the T wave, interrupting it. This is called a QT-U wave and is seen in a congenital LQTS, particularly LQTS 1 and 2, which result from a channelopathy involving the potassium channel.

The QT interval is measured from onset of QRS complex (either a Q or R wave) to end of T wave. The lead used to measure the QT interval is the one in which the T wave is most distinct and where its termination is clear. Identifying the termination of the T wave can be particularly difficult when a U wave is present. The U wave is not included in the QT measurement if it is after the T wave and distinct from and significantly smaller than the T wave. However, when the T and U waves are merged and the U wave is on top of the T wave or interrupts it, the U wave is included in the QT measurement. The QT interval needs to be corrected for heart rate, using Bazett's equation,

QTc = QT interval ÷ square root of the RR interval (in sec)

The normal corrected QT (QTc) ≤ 0.44–0.46 sec. It also should be remembered that the QT interval also includes the QRS complex. The normal QT measurement is based upon a QRS complex duration that is normal (up to 0.10 sec). Therefore, if the QRS complex duration is increased, this needs to be considered in measuring the QT interval. The duration over 0.10 sec should be subtracted from the QT interval measurement and then the QT interval corrected for heart rate.

A long QT interval may be due to:

1. Delayed repolarization in which the ST segment is long while the T wave is normal. This form of long QT interval is seen with metabolic abnormalities, particularly low calcium or magnesium.
2. Prolonged repolarization in which the ST segment is normal but the T wave is broad. This may be due to drugs, termed an acquired QT prolongation, or a genetic abnormality producing a channelopathy, called congenital LQTS). It is congenital QT prolongation that may have a prominent U wave interrupting the QT interval (QT-U wave).

continues

The LQTS is the phenotypic description of a group of disorders characterized by a prolonged QT interval in association with a characteristic arrhythmia, termed polymorphic ventricular tachycardia, which is a ventricular tachycardia with changing QRS morphologies and axis. Polymorphic ventricular tachycardia occurring in association with a long QT interval (either acquired or congenital) as measured on a sinus complex it is called torsades de pointes, or twisting of points.

The history is important for establishing the etiology for a long QT interval, *ie*, congenital and acquired. As indicated, the presence of a QT-U complex is consistent with a congenital LQTS. The history suggests that he has experienced a nonsustained ventricular tachyarrhythmia that is the etiology of his symptoms. It is most likely that he has an LQTS 1 as arrhythmic events (most often torsades de pointes) in patients with LQTS 1 are most often related to exercise or sympathetic stimulation. The QT-U wave is also consistent with LQTS 1, which is the result of a potassium channelopathy. Initial therapy for congenital QT prolongation is often a β-blocker, although an ICD is often implanted. ■

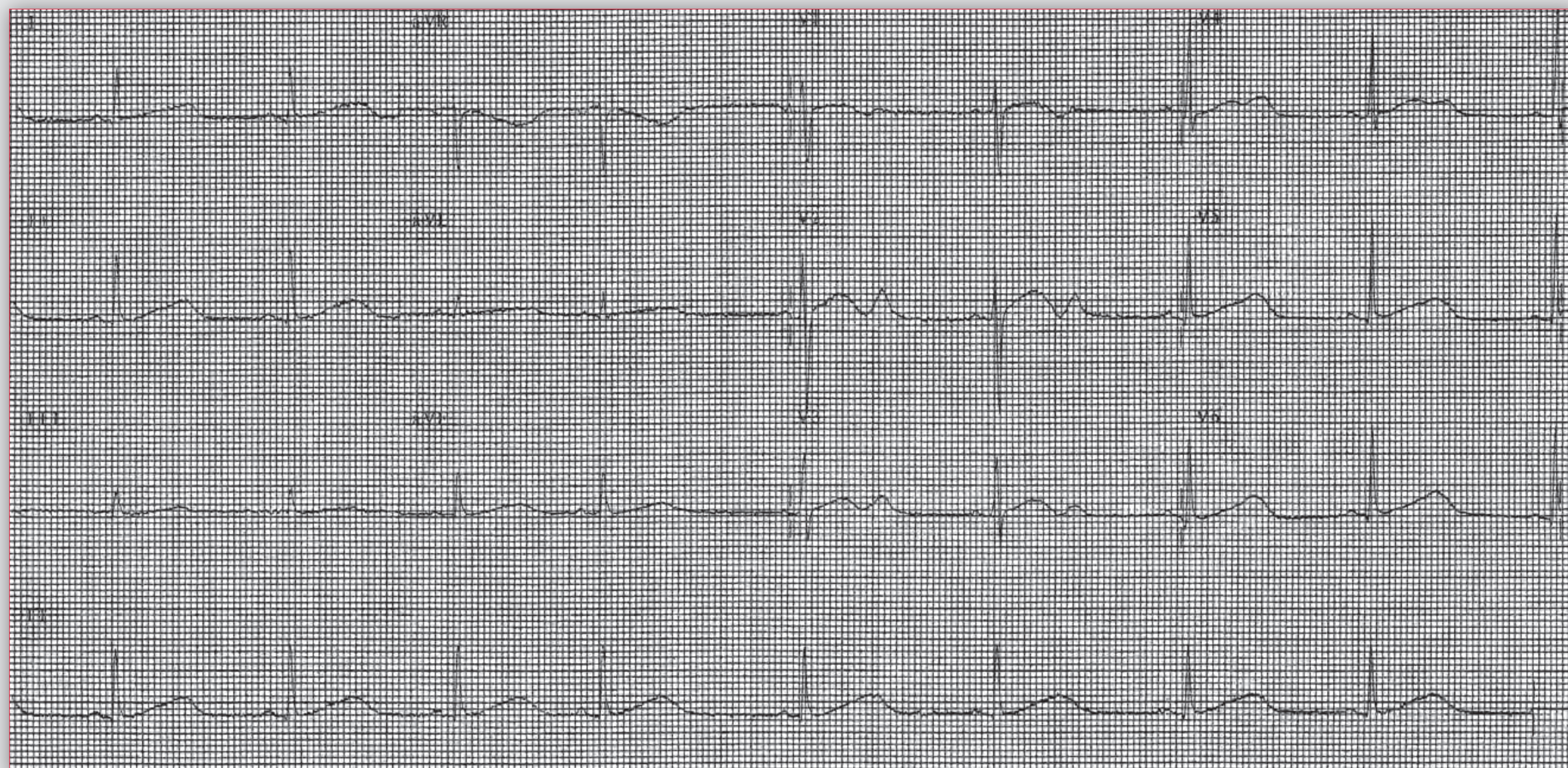
Core Case 98

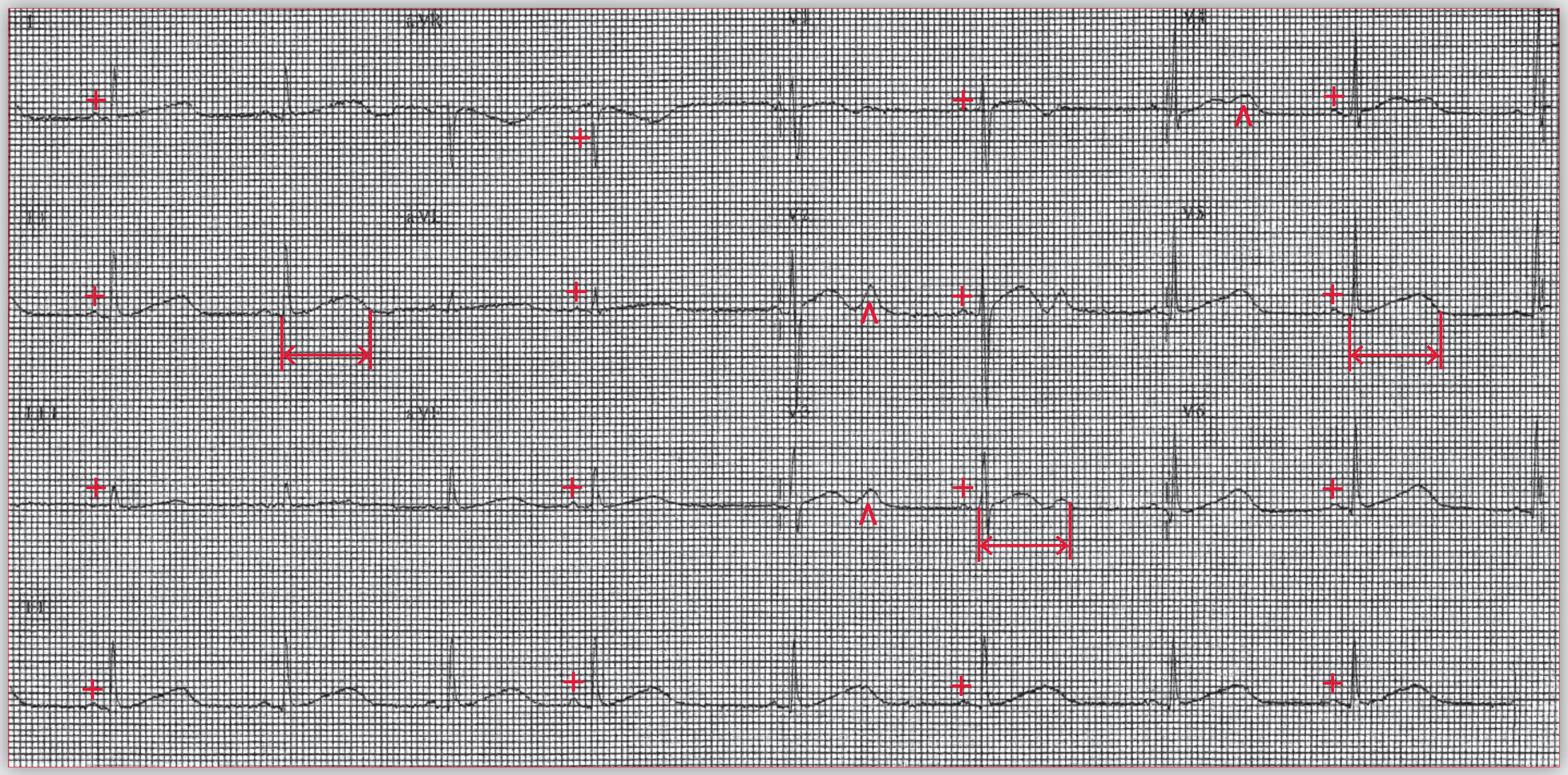
An 18-year-old female patient with anorexia nervosa is admitted to hospital with syncope. She states she has restricted her caloric intake markedly in the preceding week. She was quite lightheaded when she stood up and promptly lost consciousness. Her parents found her lying on the floor in her room and activated emergency medical services.

On admission, her exam is notable for an emaciated appearance. Her vital signs are normal. Her laboratory values are notable for reduced serum potassium and magnesium. An ECG is obtained. Based on this ECG, a more detailed history is obtained. She states that when she was younger, she had a seizure disorder for which she was treated with phenytoin. She does not think that this drug was effective. Her family history is notable for instances of unexplained sudden death across multiple generations.

What abnormalities are notable on the ECG?

In light of her history and the family history, what diagnosis can be made?





ECG 98 Analysis: Sinus arrhythmia, prolonged QT interval, QT-U wave

There is an irregularly irregular rhythm, *ie*, there is variability in the RR intervals without any pattern. The heart rate varies between 42 bpm and 66 bpm; the average rate is 54 bpm. There is a P wave (+) before each QRS complex and the PR interval is stable (0.14 sec). The P-wave morphology is uniform and positive in leads I, II, aVF, and V4–V6 and negative in lead aVR. Hence this is a normal sinus rhythm and sinus arrhythmia, which is a normal and physiologic respirophasic arrhythmia, *ie*, related to respiration. The heart rate increases with inspiration and decreases with expiration as a result of changes in autonomic inputs, *ie*, changing vagal tone.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT interval is long (↔) (600 msec), and the QTc is 570 msec. The QT interval is long as a result of a broad T waves, and hence this is prolonged repolarization or a long QT syndrome. The QT interval is best measured in leads II and V5–V6. When measured in these leads, it is apparent that the waveform seen in leads V2–V4 is a U wave (^), which interrupts the T wave and is superimposed upon it. Hence this is a QT-U wave, which is seen primarily in a congenital long QT syndrome (LQTS), particularly LQTS 1 and LQTS 2, which are the result of a potassium channelopathy (and hence the prominent U wave).

There are at least 12–13 genetic abnormalities associated with a LQTS, although the most common, accounting for almost 90% of cases, are associated with a potassium channelopathy, *ie*, LQTS 1 or LQTS 2. The most important complication of a LQTS is a life-threatening ventricular tachyarrhythmia, primarily polymorphic ventricular tachycardia (termed torsades de pointes) or ventricular fibrillation. In this case, the patient began having symptoms as a child, presenting with seizures. This is a not uncommon presentation for those with a LQTS as the seizures may be the manifestation of a serious ventricular tachyarrhythmias. In this case, the long QT interval was possibly exposed by electrolyte abnormalities. The occurrence of a syncopal episode, likely the result of a ventricular arrhythmia, was possibly provoked by sympathetic activation related to anorexia and possible hypotension.

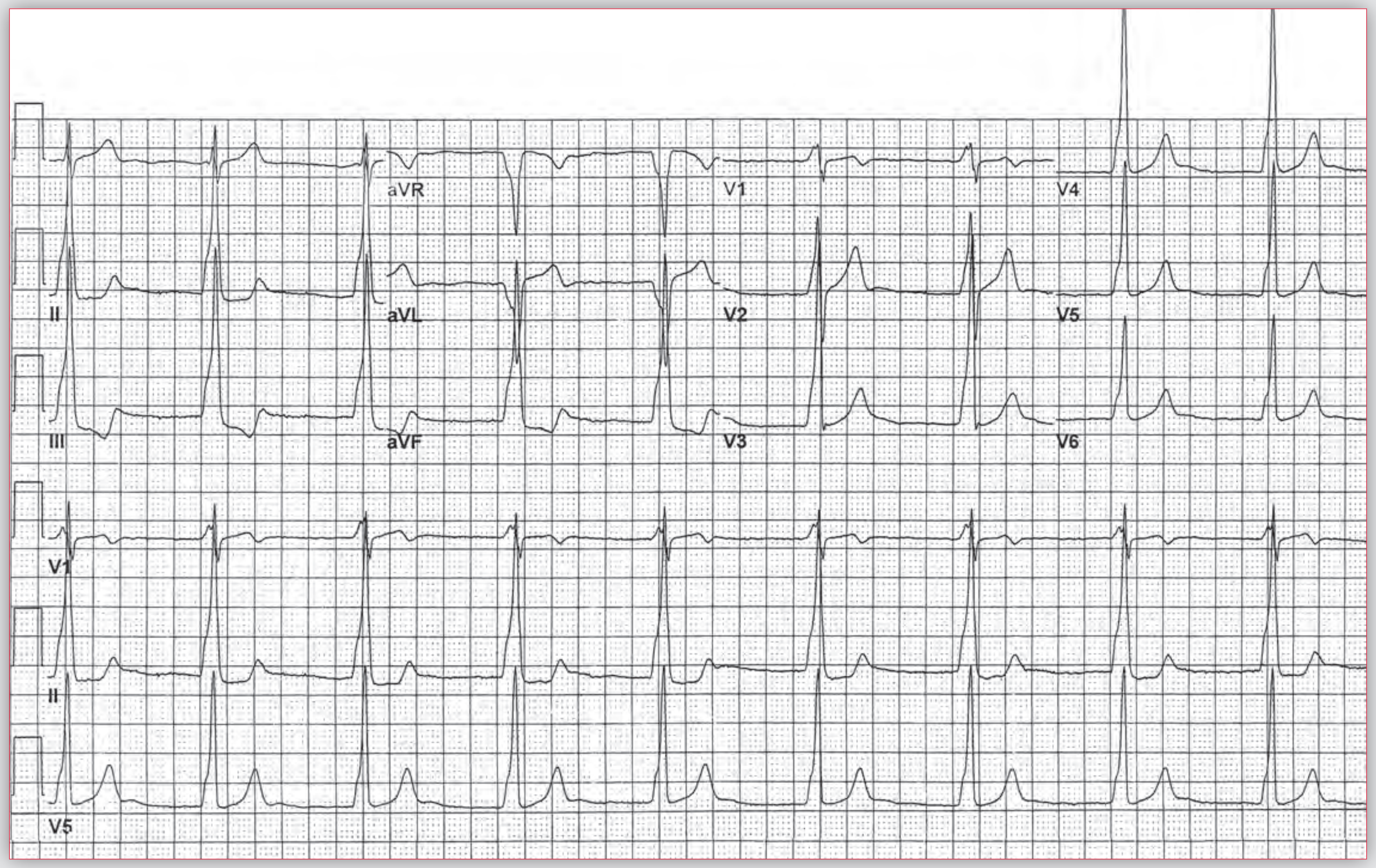
Recommended therapy for a LQTS includes:

1. β-blocker therapy is recommended for patients with QT prolongation, even those receiving an implantable cardioverter-defibrillator (ICD).
2. ICD implantation is recommended for survivors of a cardiac arrest.
3. ICD implantation is suggested for patients who experience sustained VT and/or a syncopal event consistent with a tachyarrhythmia while on β-blocker therapy.
4. ICD implantation combined with β-blocker therapy is suggested for patients at an increased risk of sudden cardiac death, such as those with a family history or in those where compliance with β-blocker therapy is a consideration. ■

Core Case 99

A 30-year-old man presents to his physician with complaints of palpitations. He is without symptoms at the time of the office visit. He has no medical problems and is not on any medication. His physical examination is unremarkable. An ECG is obtained (ECG 99A) that alarms the physician. As a result, he requests a repeat ECG (ECG 99B).

ECG 99A

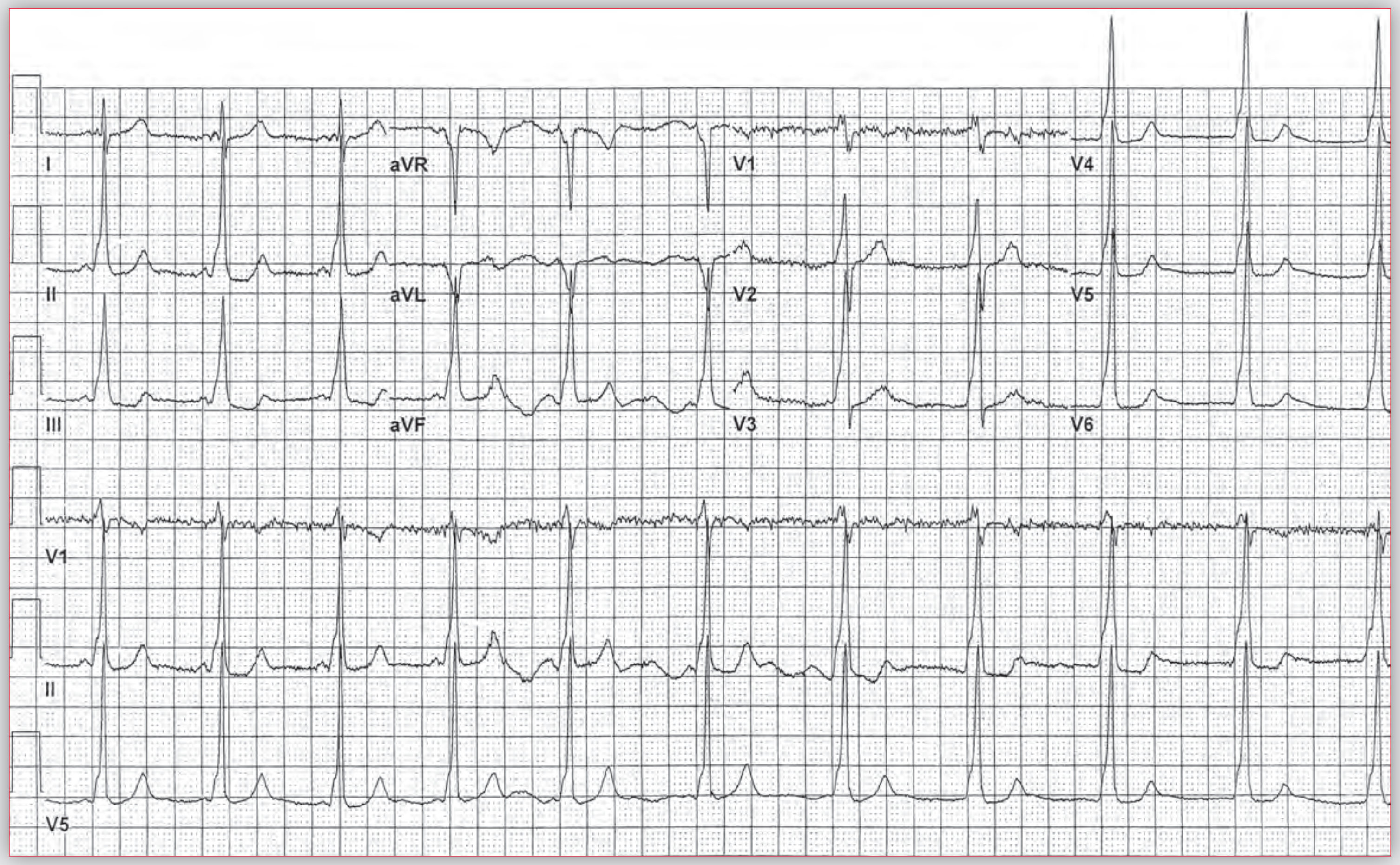


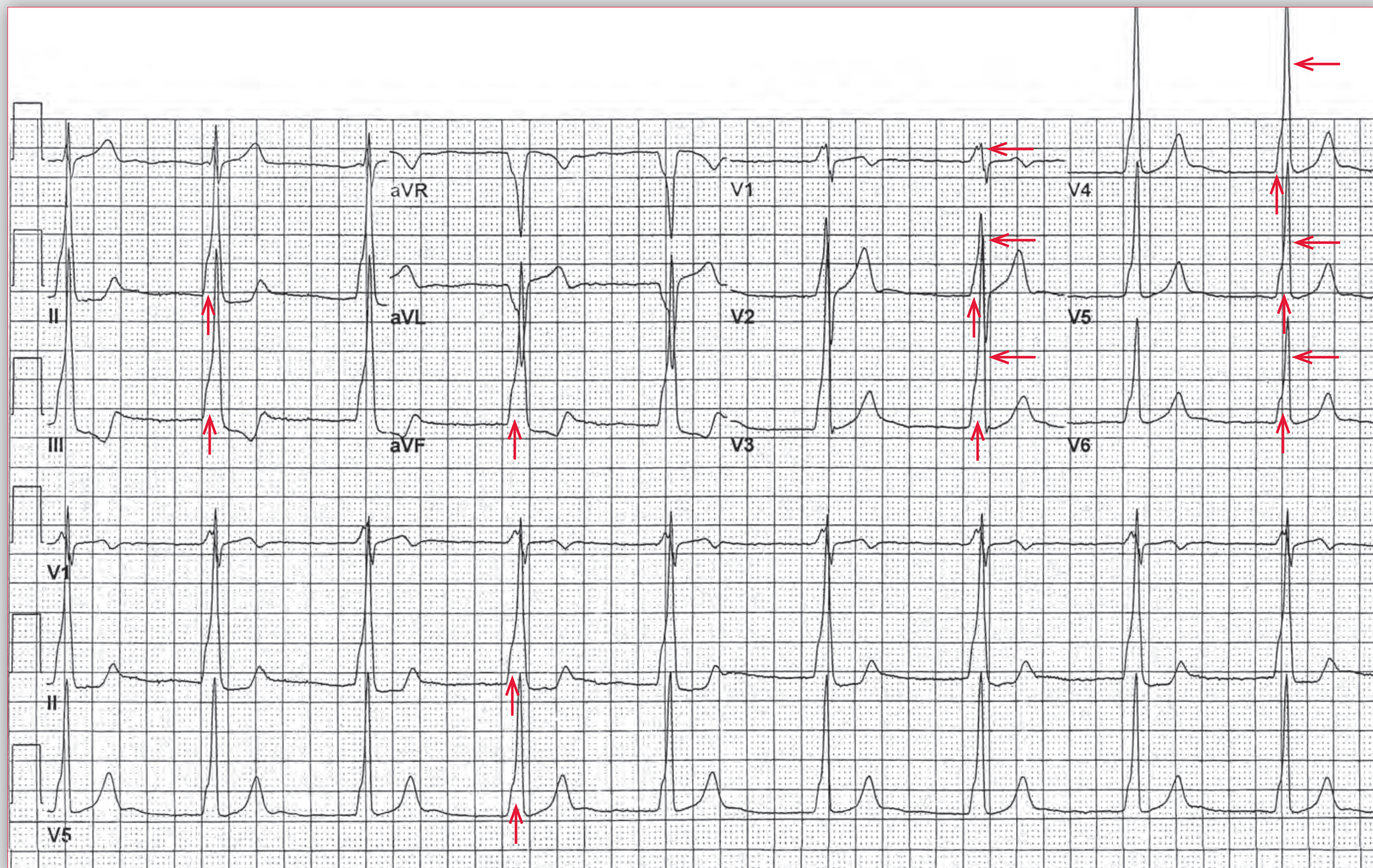
What does ECG 99A show that causes concern?

What is the rhythm?

What abnormality is seen on ECG 99B?

ECG 99B





ECG 99A Analysis: Wide complex rhythm (ventricular versus preexcitation)

ECG 99A shows there is a regular rhythm with a rate of 50 bpm. No P waves are seen before or after any QRS complex. The QRS complex duration is increased (0.18 sec). There appears to be a slurred upstroke of the QRS complex (↑), accounting for the widening of the QRS complex at the base, while the top of the QRS complex is narrower. The QT/QTc intervals are prolonged (500/460 msec) but they are normal when the prolonged QRS complex duration is considered (420/390 msec). The QRS complex has a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). Noted is positive concordance, *ie*, tall R waves across the precordium from V1–V6 (←). The QRS complex duration and morphology are consistent with either a ventricular complex or a Wolff-Parkinson-White (WPW) pattern. However, the absence of P wave makes WPW a less likely diagnosis. The accessory pathway begins in the atrium, and a WPW pattern on the ECG can only be seen if there is initial atrial activity. While a slurred upstroke of the QRS complex, representing direct myocardial activation, may be seen with either a ventricular complex or a WPW pattern, a slurred upstroke primarily altering the initial portion of the QRS complex upstroke is more characteristic of a WPW pattern. In this case, the slurred upstroke is known as a delta wave. This indicates that initial ventricular activation is directly through the ventricular myocardium and not the normal His-Purkinje system, while ventricular activation thereafter is via the normal His-Purkinje pathway. In contrast, all of ventricular activation is abnormal in a ventricular rhythm and the whole QRS complex would be wide and abnormal.

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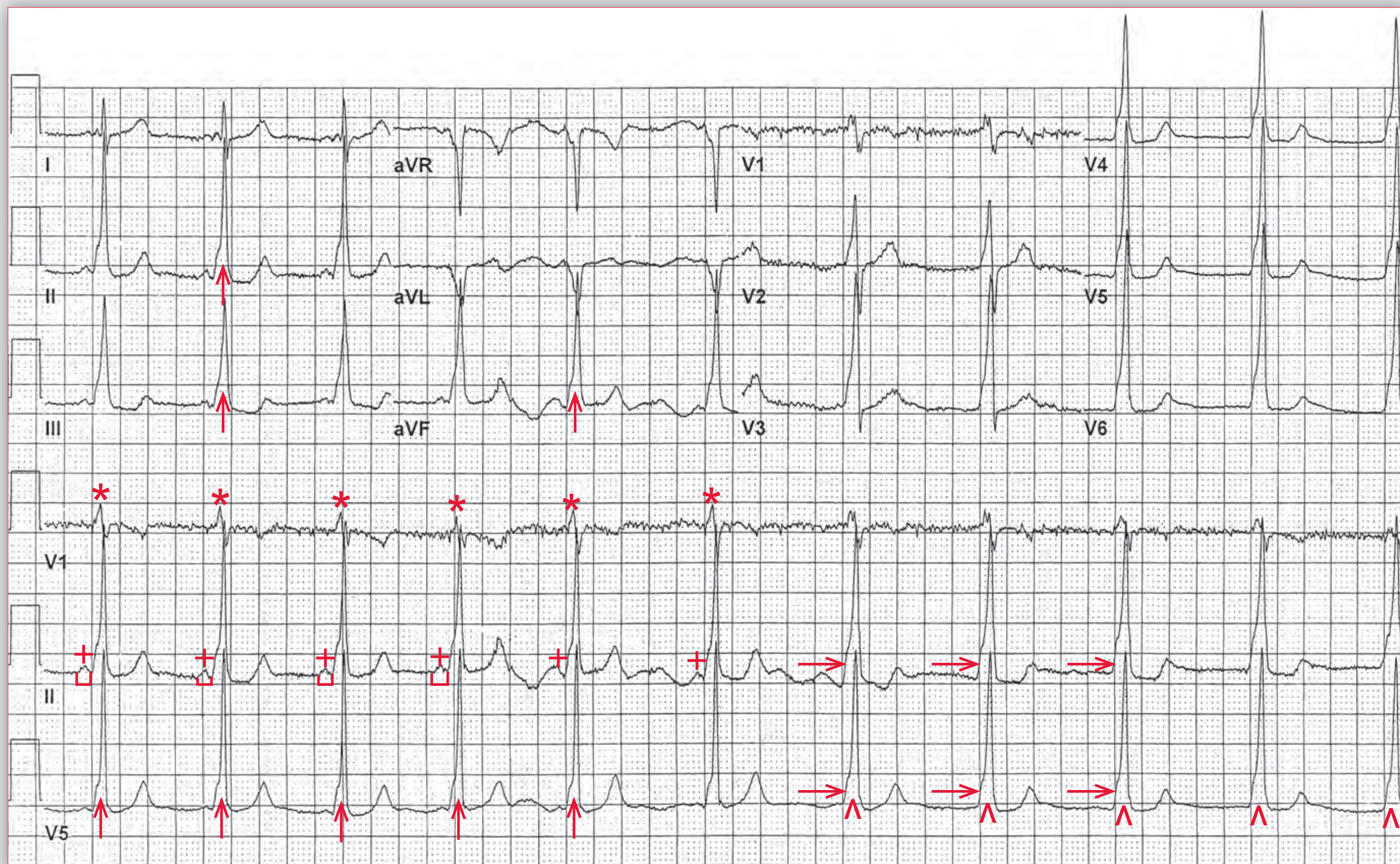


ECG 99B Analysis: Normal sinus rhythm, Wolff-Parkinson-White (WPW) pattern, concertina effect

ECG 99B is from the same patient as ECG 99A. There is a regular rhythm at a rate of 60 bpm. The QRS complex morphology and axis are identical to what is seen in ECG 99A. There is a slurred upstroke of the QRS complex as was seen in ECG 99A. The first six QRS complexes (*) have a prolonged QRS complex of 0.16 sec. This is due to a slurred upstroke of the initial portion of the QRS complex (↑), while the remainder of the QRS complex is narrower There are P waves seen before these first six QRS complexes (+). The P waves are positive in leads I, II, aVF, and V5. Hence this is a sinus rhythm. The PR interval is constant but short (0.12 sec) (⊔). In addition, the QRS complex duration of the first six QRS complexes is slightly shorter (0.16 sec) than was seen in ECG 99A. The short PR interval and prolonged QRS complex is characteristic of a WPW pattern. The slurred upstroke of the initial portion of the QRS complex is termed a delta wave.

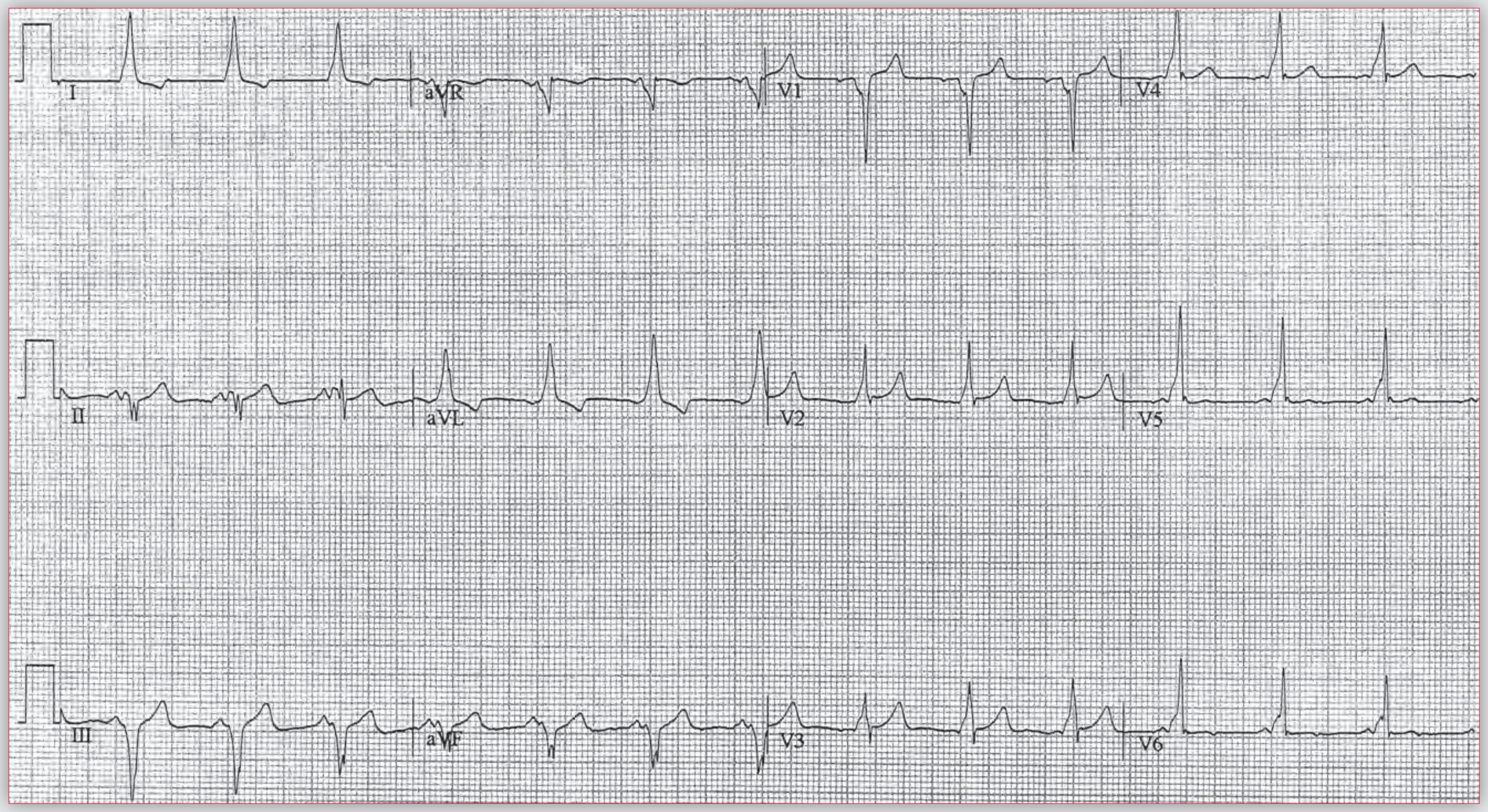
The QRS complex morphology of the remaining QRS complexes (complex 7–11) (^) is the same, but they have a wider duration (0.18 sec) and a more prominent slurred upstroke or delta wave. These complexes are identical to those seen in ECG 99A. In addition, there are no P waves before any of these QRS complexes, similar to what was seen in ECG 99A. Nevertheless, since all of the QRS complexes are identical, except for a difference in duration, complexes 7–11 also show a WPW morphology. The PR interval is shorter than what was seen in complexes 1–6 and the P wave is likely simultaneous with the delta wave and hence not seen. With the shortening of the PR interval and disappearance of the P wave, the delta wave is broader and more prominent (→), accounting for the increased QRS complex duration. The changing PR interval and delta wave width seen in WPW are termed the concertina effect. There is a Q wave in lead aVL and a positive delta wave in lead V1. This is consistent with a left lateral bypass tract. The Q wave in aVL is termed a pseudo lateral myocardial infarction. With WPW, abnormalities of the ventricular myocardium cannot be reliably interpreted since initial ventricular activation is direct, via the accessory pathway and not the normal His-Purkinje system.

The QRS complex in WPW is a fusion complex, representing ventricular activation via the accessory pathway as well as via the AV node–His-Purkinje system. The PR interval duration, the prominence and duration of the delta wave and duration of the QRS complex (*ie*, the amount of preexcitation), depends upon the balance of conduction velocity between these two pathways. This is primarily related to AV nodal conduction velocity since conduction through the accessory pathway, which is modified His-Purkinje tissue, is all or none and the conduction velocity through this pathway does not vary. The conduction velocity through the AV node is variable. Hence if there is a delay of conduction through the AV node, more of ventricular activation originates from the accessory pathway, resulting in a shorter PR interval, more pronounced delta wave, and wider QRS complex. If AV nodal conduction is faster, less ventricular activation is via the accessory pathway as there is more of ventricular activation via the normal pathway. In this situation, the PR interval is longer, the delta wave less pronounced, and the QRS complex narrower. Any changes in autonomic input into the AV node can affect the conduction velocity through this structure and can alter the degree of preexcitation. ■

Core Case 100

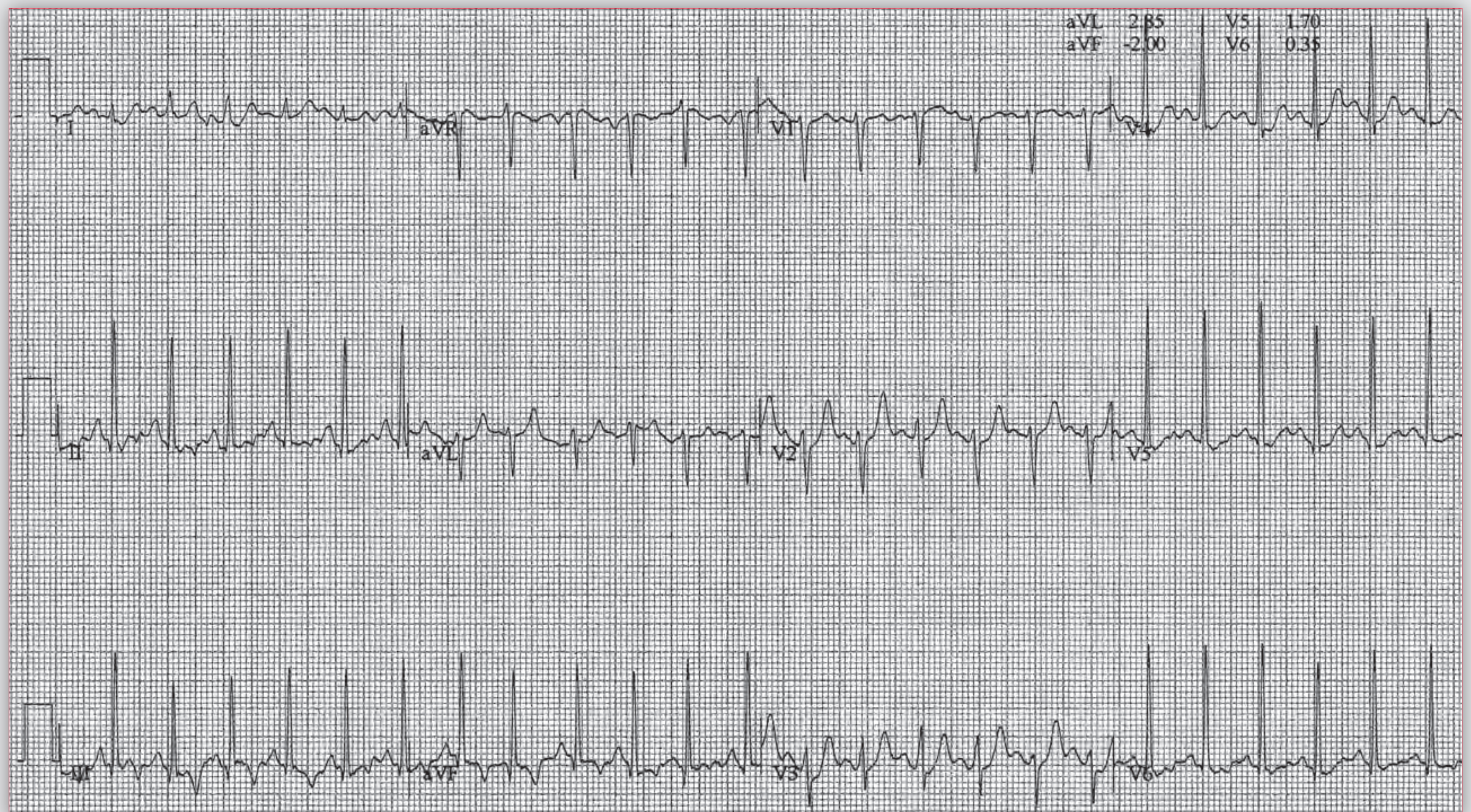
A 23-year-old man is seen for a routine pre-employment physical examination. He denies all cardiac symptoms. An ECG (ECG 100A) is obtained and his physician becomes

ECG 100A



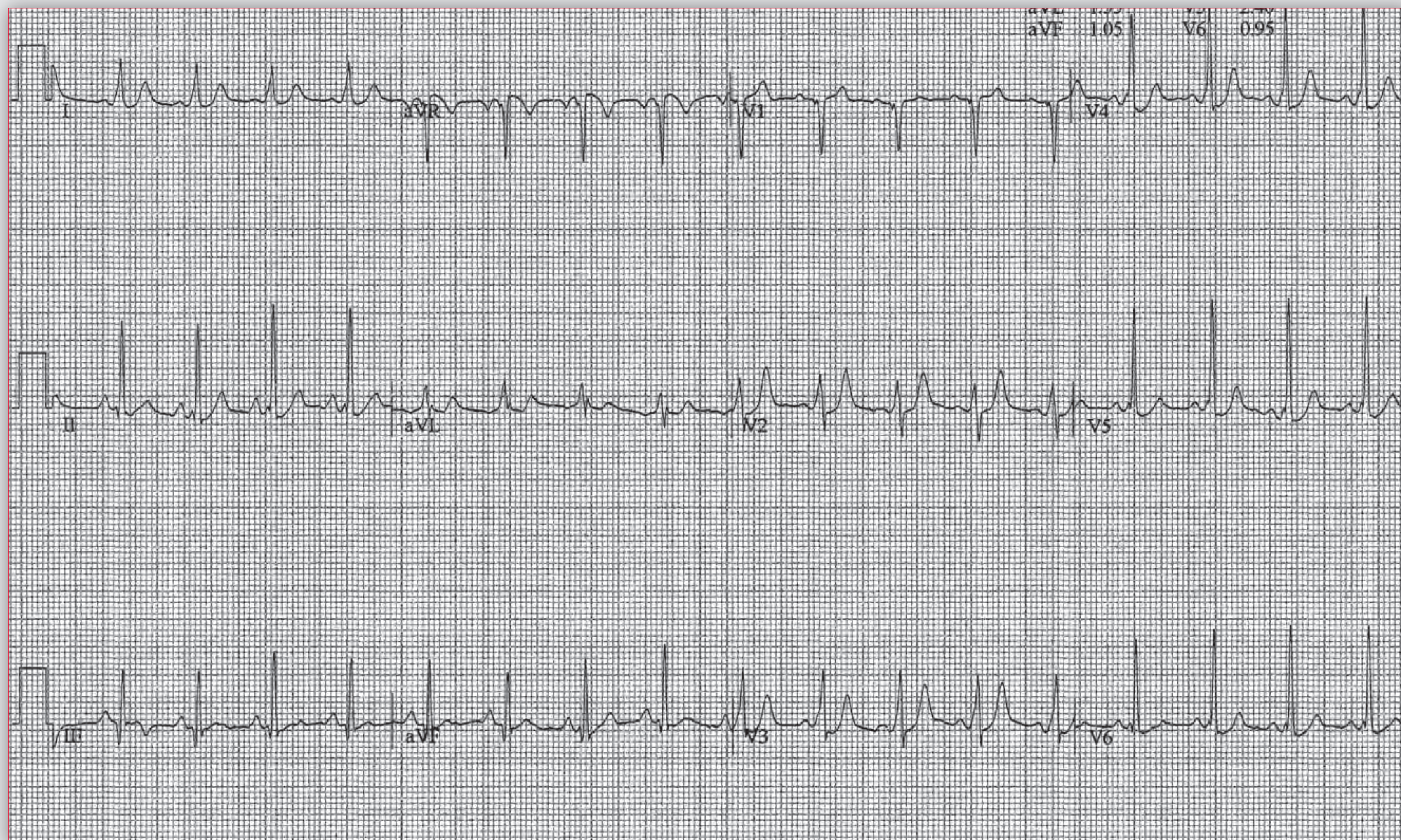
concerned. As a result, he sends the patient for an exercise test. ECG 100B is from 6 minutes of the exercise test and ECG 100C is during early recovery.

ECG 100B



Core Case 100

ECG 100C



Core Case 100

What are the findings on these ECGs?

What further evaluation or therapy would be necessary?

ECG 100A Analysis: Normal sinus rhythm, Wolff-Parkinson-White (WPW) pattern, pseudo inferior wall myocardial infarction

ECG 100A shows there is a regular rhythm with a rate of 82 bpm. There is a P wave (**+**) before each QRS complex with a stable but short PR interval (0.10 sec). The P wave is positive in leads II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is increased (0.12 sec), and it can be seen that the widening is primarily at the base of the QRS complex, as a result of a slurred upstroke (↑); the peak of the QRS complex is narrow. This slurred upstroke is known as a "delta" wave, and along with the short PR interval is characteristic of a WPW pattern. The delta wave is due to initial ventricular activation occurring as a result impulse conduction through the accessory pathway, resulting in direct myocardial activation that is early resulting from the bypass of the AV node (hence the short PR interval) and slow (hence the delta wave). The QT/QTc intervals are prolonged (380/445 msec and 360/420 msec when the prolonged QRS complex duration is considered).

Noted are Q waves in leads II, III, and aVF (←). However, as indicated, in WPW ventricular activation is not initiated through the normal His-Purkinje pathway, but rather by direct myocardial activation via the accessory pathway. Therefore, similar to a left bundle branch block, paced rhythm or a ventricular rhythm, abnormalities of the left ventricular myocardium cannot be reliable diagnosed. Thus the inferior Q waves are not the result of an inferior wall myocardial infarction, but rather they reflect the delta wave, and this is termed a pseudo inferior wall infarction pattern. A pseudo inferior wall infarction pattern is associated with a posteroseptal bypass tract. As the delta wave is negative in lead V1, meaning that the impulse is going from anterior (right ventricle) to posterior (left ventricle), the accessory pathway is right-sided.

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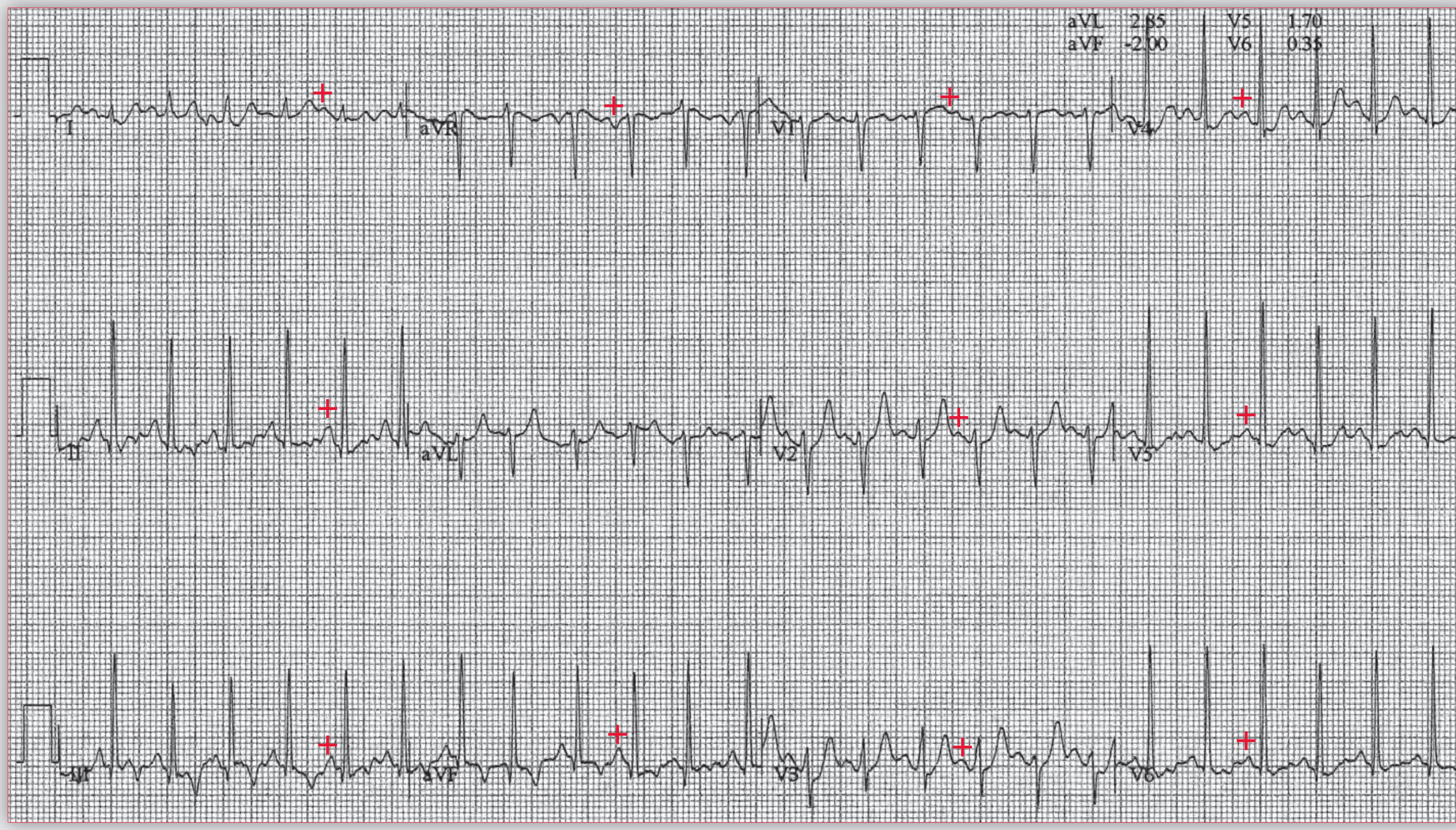
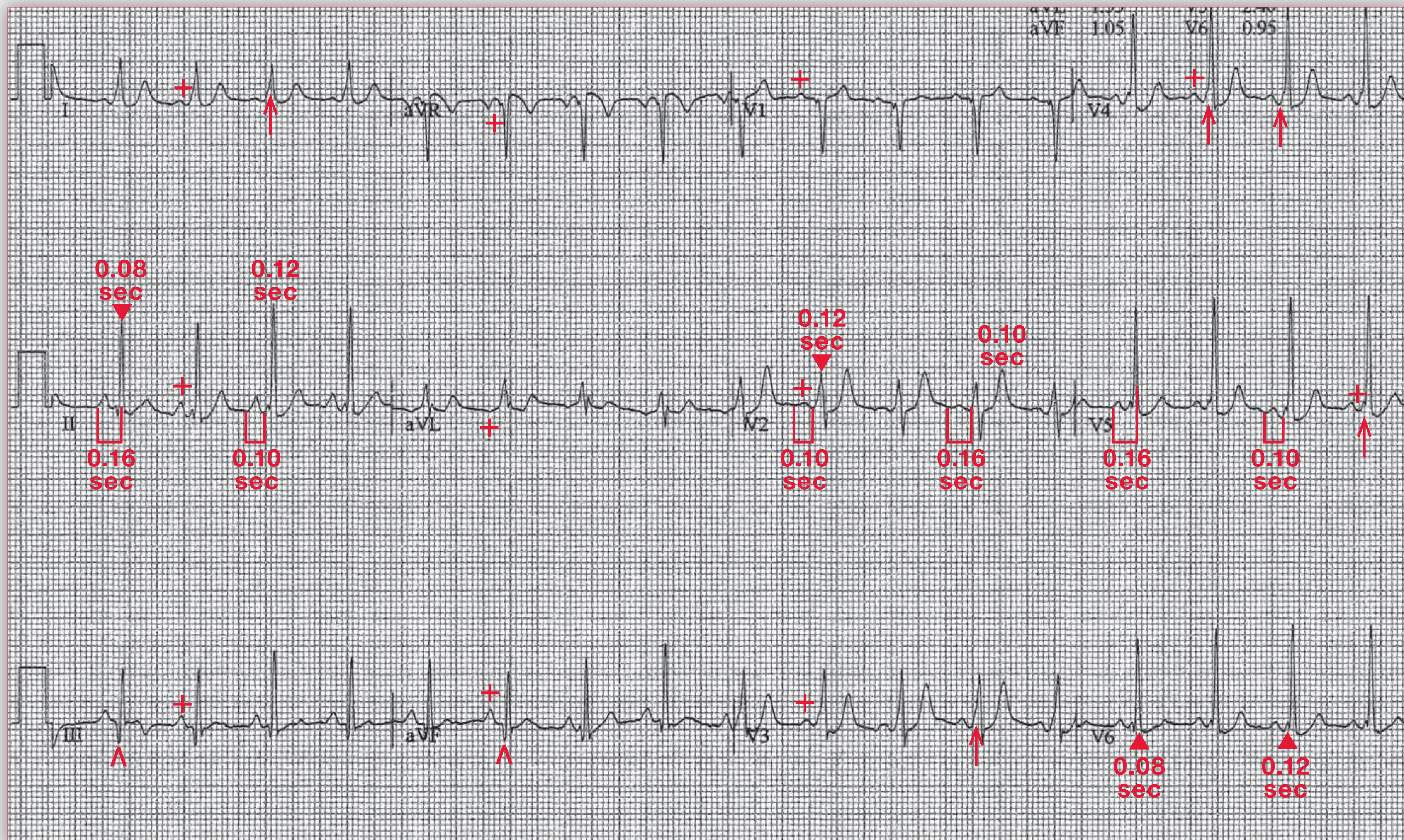


ECG 100B Analysis: Sinus tachycardia, normal QRS complex (loss of WPW pattern)

ECG 100B is from the same patient as ECG 100A and was obtained 6 minutes into an exercise test. There is a regular rhythm at a rate of 150 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia, the result of exercise. The QRS complex duration is normal (0.08 sec) and no delta wave is seen. In addition there are no inferior Q waves present. The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/380 msec). Therefore, with exercise there has been resolution of the WPW pattern. This is due to the fact that the WPW QRS complex is the result of fusion between early impulse conduction through the accessory pathway and slightly later impulse conduction occurring through the normal AV node–His-Purkinje system. If conduction through the AV node is slow, most of ventricular activation results from impulse conduction through the accessory pathway. In this situation, the PR interval is shorter and the delta wave more prominent. If AV nodal conduction is enhanced and faster, less of myocardial activation is the result of impulse conduction through the accessory pathway, and hence the PR interval is longer and the delta wave less pronounced. The disappearance of the delta wave means that all of ventricular activation is via the normal AV node–His-Purkinje system. Since this occurs with exercise, there is sympathetic enhancement of conduction through the AV node and all of ventricular activation is by this pathway. As accessory pathway tissue is similar to Purkinje tissue, its conduction is not altered by sympathetic tone or catecholamines. The disappearance of the delta wave with exercise implies that the refractoriness of the accessory pathway, which does not change, is longer than the AV nodal refractoriness (which is shortened by sympathetic stimulation or catecholamines). Hence at the faster rates, impulse conduction is preferentially through the AV node. In general, patients who lose the WPW pattern with exercise have a much lower risk of experiencing sudden death, as the accessory pathway is not capable of conducting impulses at very rapid rates. Sudden death in WPW is the result of atrial fibrillation and rapid conduction through the accessory pathway, causing a very fast ventricular response (often well above 350 bpm), which can result in ventricular fibrillation even in a structurally normal heart.

continues



ECG 100C Analysis: Sinus tachycardia, intermittent WPW pattern

ECG 100C was obtained during recovery. There is a regular rhythm at a rate of 112 bpm. There are P waves before each QRS complex (+) with a PR interval that varies between 0.10 to 0.16 sec. The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complex duration is also variable between 0.08 to 0.12 sec. This is due to a delta wave (↑) that also varies in its width or prominence. However, it is less obvious than that seen in ECG 100A. In addition, small Q waves (^) can be seen in leads III and aVF. Hence as the heart rate slows, due to a decrease in sympathetic stimulation, there is slower impulse conduction through the AV node and more of left ventricular activation is due to impulse conduction via the accessory pathway. In addition there is variability in the QRS width (▼, ▲) (and delta wave prominence) and PR intervals (⊔), as is obvious in leads II, aVF, V2, and V5. This is the result of variability in the rate of AV nodal conduction and the degree of fusion between conduction via the AV node–His-Purkinje system and conduction via the accessory pathway. Since the WPW pattern represents fusion between impulses conducted through these two pathways, any changes in the rate of AV nodal conduction velocity will affect the amount of fusion, *ie*, the extent of myocardium activated via the accessory pathway. The changes in PR interval and QRS complex width (due to delta wave prominence) is called the concertina effect. ■

Notes

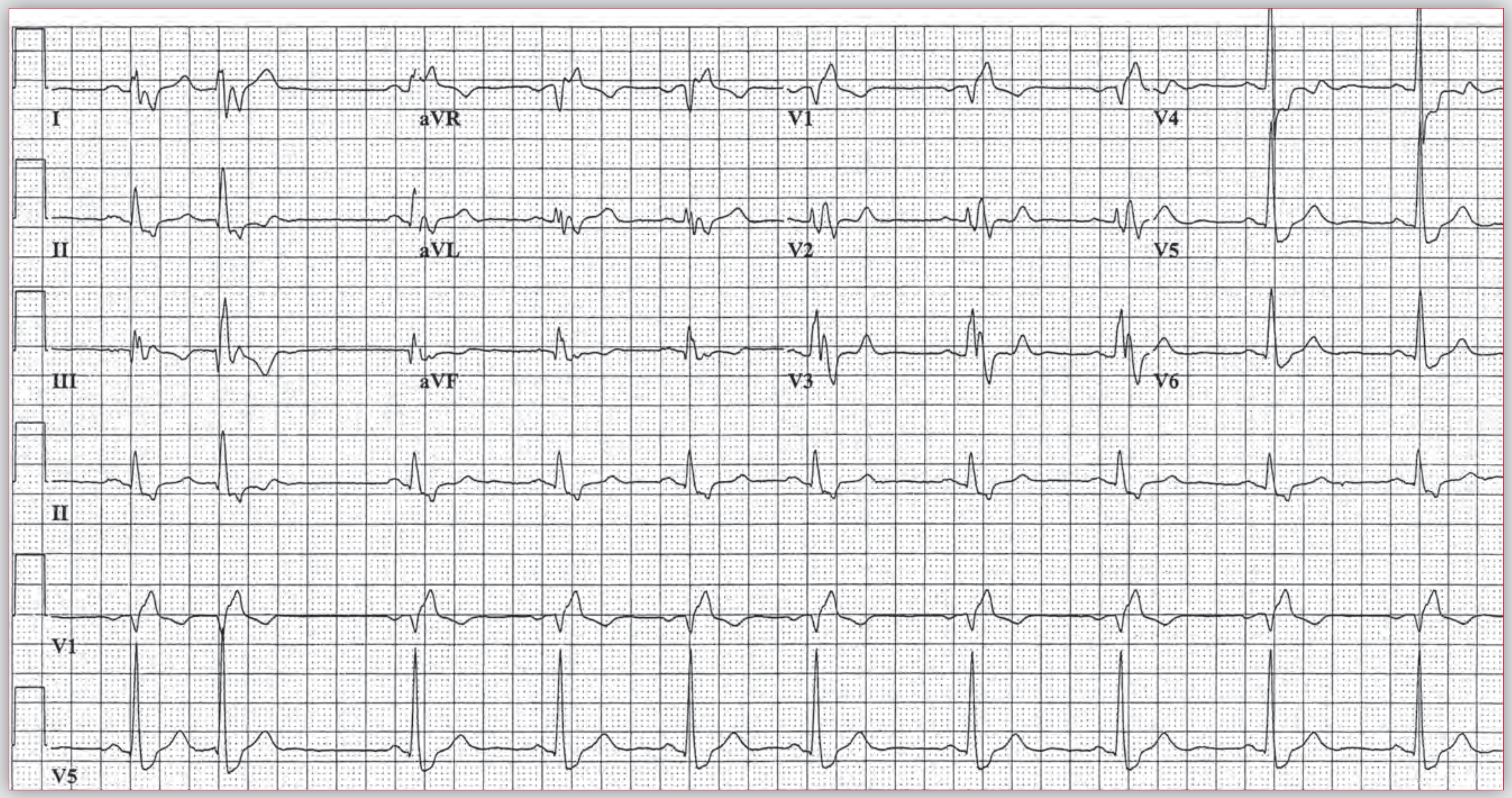
Core Case 101

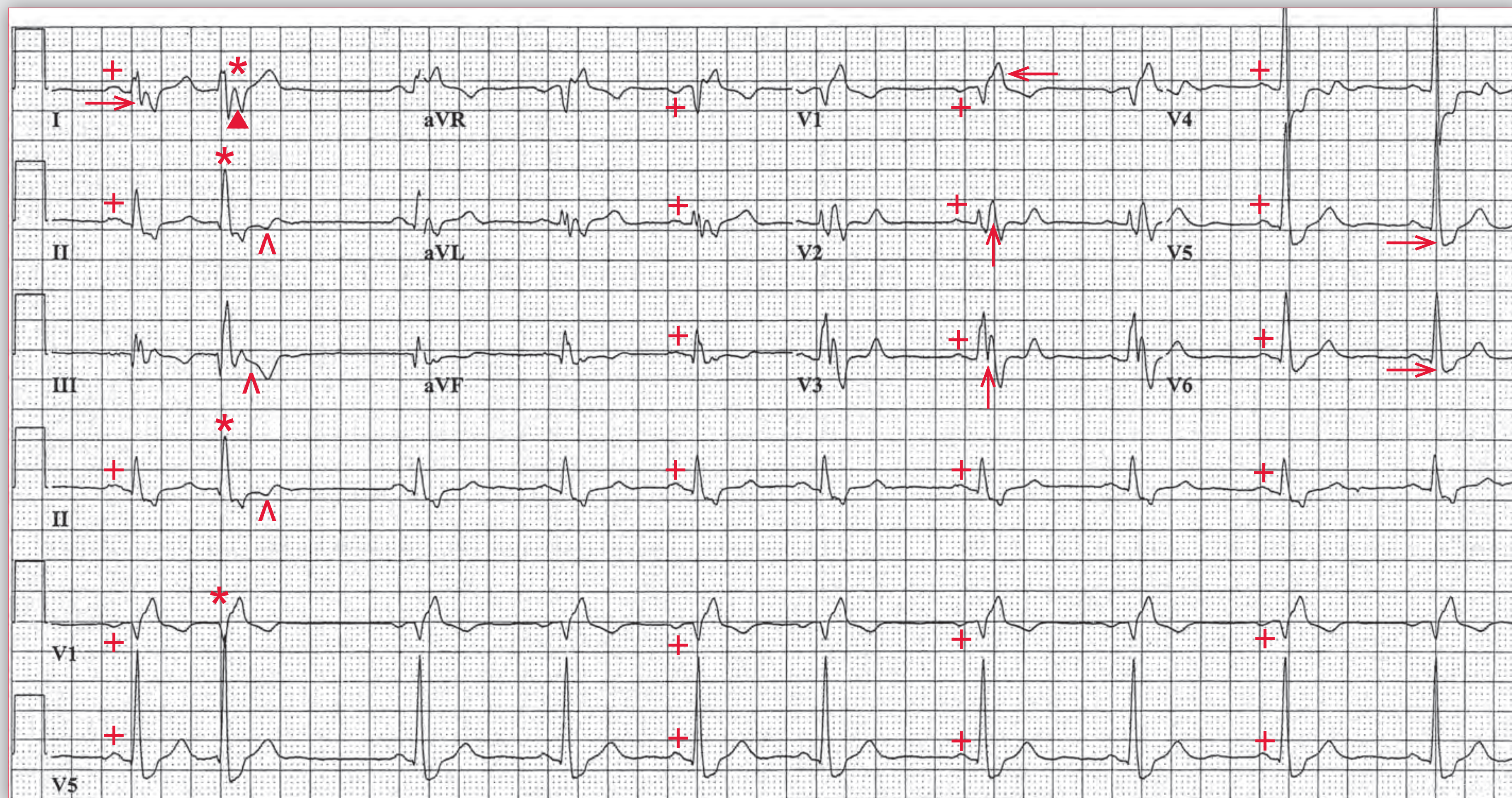
A 46-year-old man is referred to a cardiologist because of palpitations. He states that he has had a murmur since childhood, but has never had any cardiac symptoms. He has never had any evaluation for the murmur. On physical examination, there is a loud holosystolic murmur, heard best to the right of the sternum. It increases in intensity with inspiration. Examination of the neck demonstrates a prominent “V” wave, although the neck veins are not distended. An ECG is obtained.

Is the ECG normal?

If not, what abnormalities are noted?

What is the likely etiology for the murmur?





ECG 101 Analysis: Sinus arrhythmia, premature junctional complex, right bundle branch block (RBBB), "splintered" QRS complex (RSR′S′ in leads V2–V3), Ebstein's anomaly

There is an irregularly irregular rhythm at an average rate of 60 bpm. There is a P wave (+) before each of the QRS complexes (except for complex 2) with a stable PR interval (0.16 sec). The P-wave morphology is constant and positive in leads I, II, aVF, and V4–V6. Hence this is a sinus arrhythmia. The second QRS complex (*) is premature. It has a morphology that is the same as the sinus complexes, although the amplitude is taller in leads II, III, and V5. There is no P wave before this complex, but there is a P wave after the QRS complex (^), which is a negative deflection within the ST segment. Hence this is a premature junctional complex. Premature junctional complexes are supraventricular, but without a preceding P wave; they may have a retrograde P wave after the QRS complex. The QRS complexes commonly have a slightly different amplitude and axis compared to the sinus complexes due to the fact that the impulse originates from an ectopic focus within the AV junction and enters the bundle of His at a slightly different location compared to the impulse coming through the AV node. Hence the conduction pathway or tract through the His-Purkinje system may be slightly different.

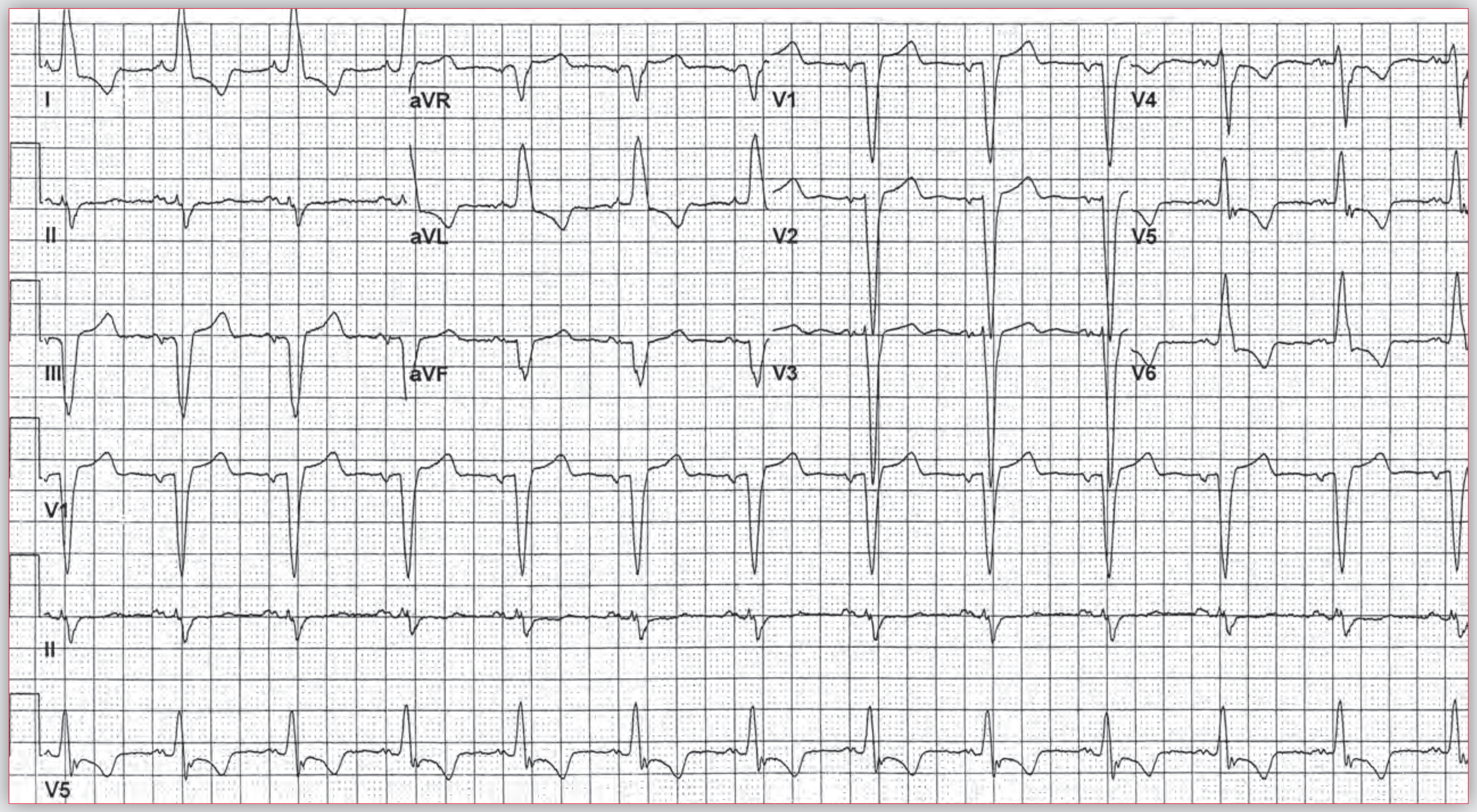
The QRS complex duration is increased (0.20 sec) and the morphology is typical for a RBBB with a RSR′ complex in lead V1 (←) and broad S waves in leads I, V5–V6 (→). However, the QRS width is wider than usual for a typical RBBB. In addition, there appears to be significant widening and notching of the S wave in lead I (▲). The QRS complexes in leads V2–V3 have an RSR′S′ morphology (↑). These features suggest a marked right ventricular conduction abnormality. This is termed a "splintered" QRS complex and is often seen in Ebstein's anomaly. An additional feature, not seen on this ECG, are "Himalayan" P waves, which are the result of significant right atrial hypertrophy.

The murmur that is heard is tricuspid regurgitation that is typically a holosystolic murmur (sounding like mitral regurgitation) heard on the right side of the sternum. The change with respiration (*ie*, increased intensity with inspiration and decrease with expiration) results for changing venous return and right ventricular filling, which produces a change in the amount of regurgitation. The prominent V wave in the jugular pulse reflects the tricuspid regurgitation. Palpitations in Ebstein's anomaly are due to atrial tachyarrhythmias. However, about 20%–30% of patients have an associated accessory pathway or Wolff-Parkinson-White pattern. In these patients, an atrioventricular nodal reentrant tachycardia (AVNRT) may occur. ■

Core Case 102

A 62-year-old man with known hypertension and hyperlipidemia presents to the emergency department with the acute onset of substernal chest pressure and nausea. Symptoms have been present for about 1 hour. His prior ECG showed a left bundle

ECG 102A

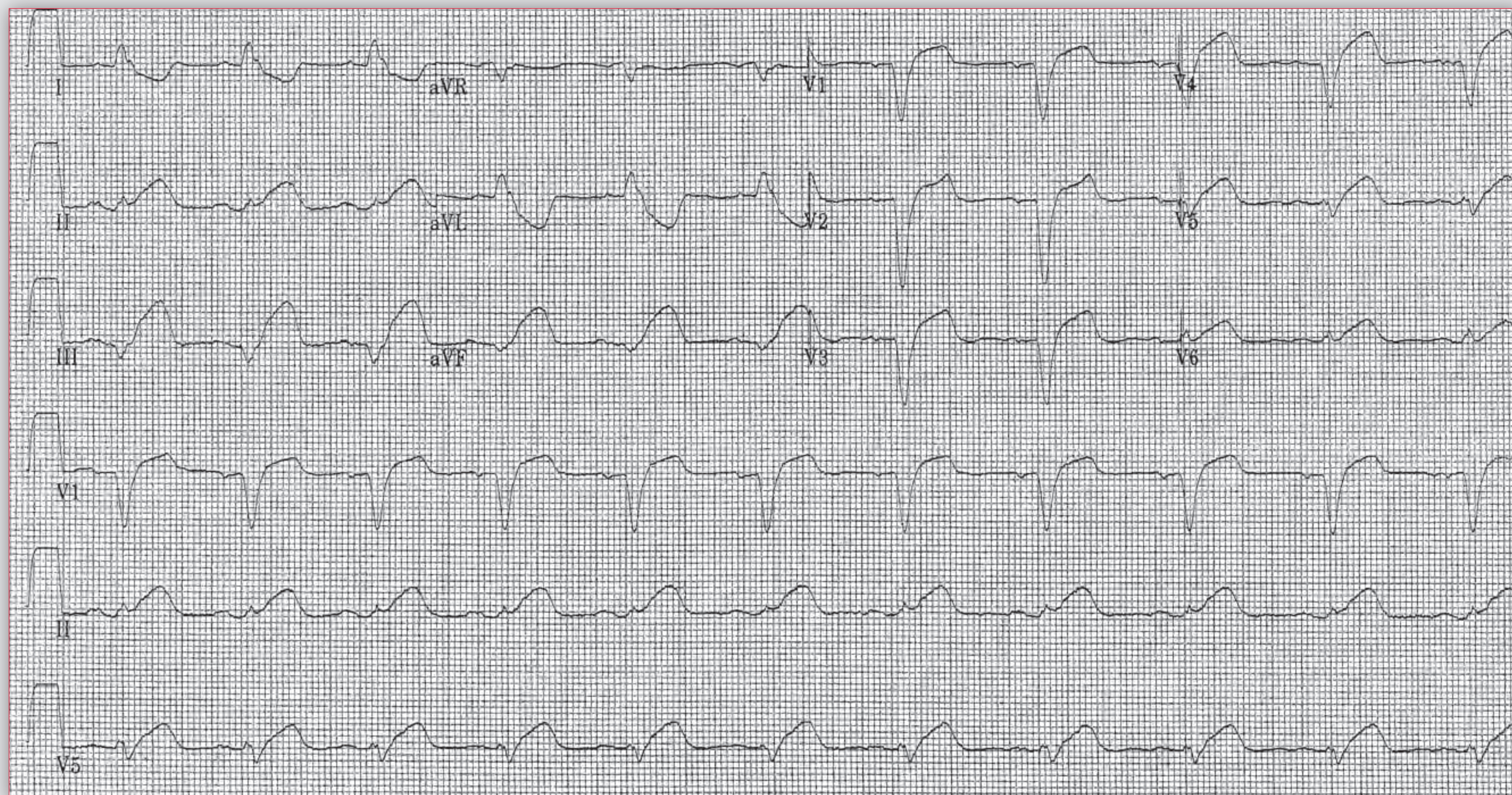


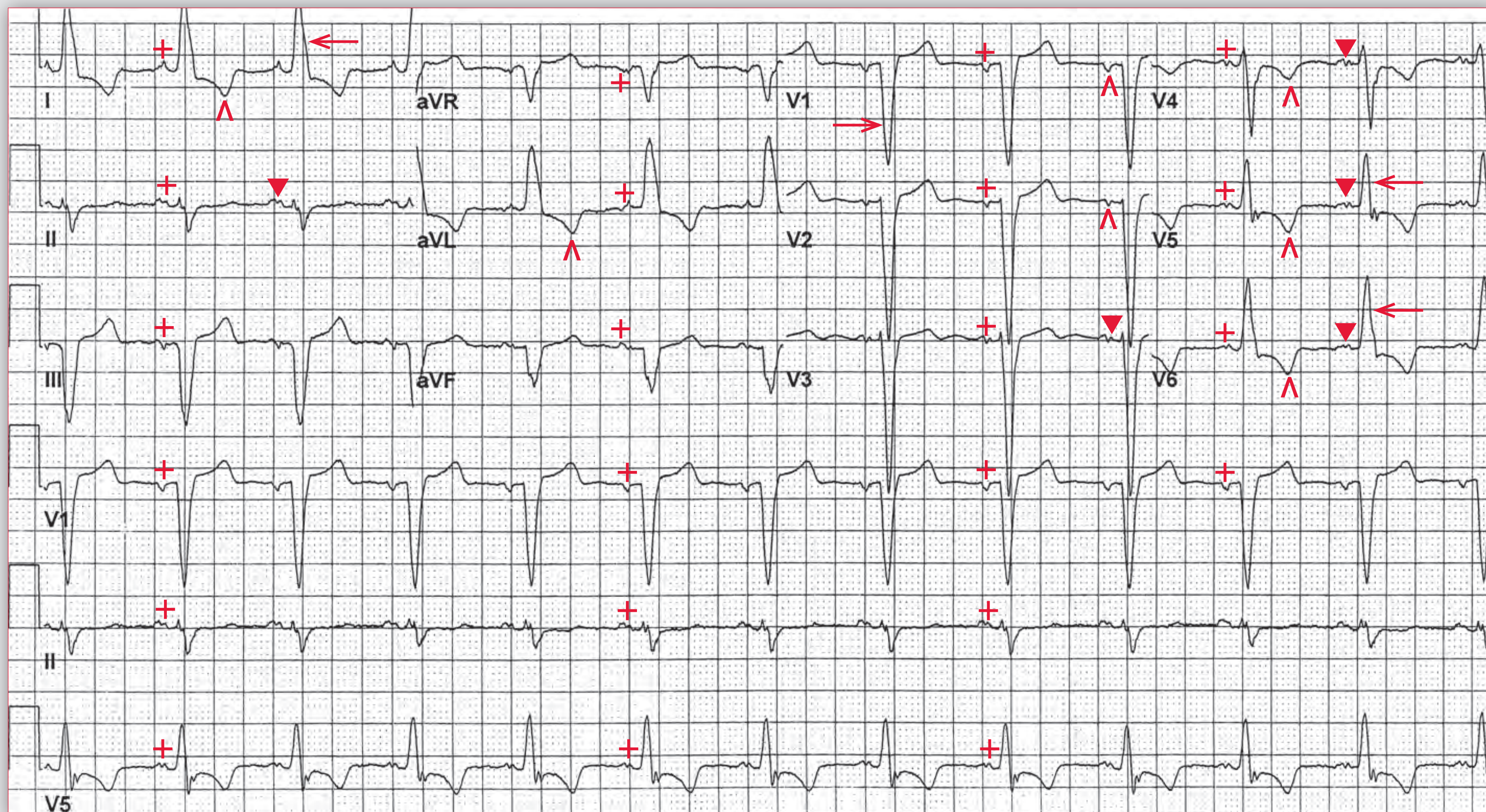
branch block (ECG 102A). The emergency department physician obtained an ECG (ECG 102B) but does not note any major abnormality and feels that the diagnosis of an acute myocardial infarction will depend upon the cardiac biomarkers.

Does the ECG show any abnormality of concern?

What is the appropriate therapy for this patient?

ECG 102B





ECG 102A Analysis: Normal sinus rhythm, left axis, left bundle branch block (LBBB), left atrial hypertrophy (or abnormality)

ECG 102A shows there is a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The P waves are, however, broad and prominently notched in leads II and V3–V6 (▼). This P wave morphology is termed "P-mitrale" and is consistent with left atrial hypertrophy (or left atrial abnormality). In addition, there is a negative P wave (^) in leads V1–V2, which is another feature seen with left atrial hypertrophy.

The QRS complex duration is prolonged (0.14 sec) and the morphology is typical for a LBBB with a broad R wave in leads I and V5–V6 (←) and a QS complex in lead V1 (→). Associated with the LBBB are non-specific ST-T wave changes (^). The QT/QTc intervals are prolonged (410/460 msec) but are normal when the prolonged QRS complex duration is considered (370/415 msec). The axis is extremely leftward between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF).

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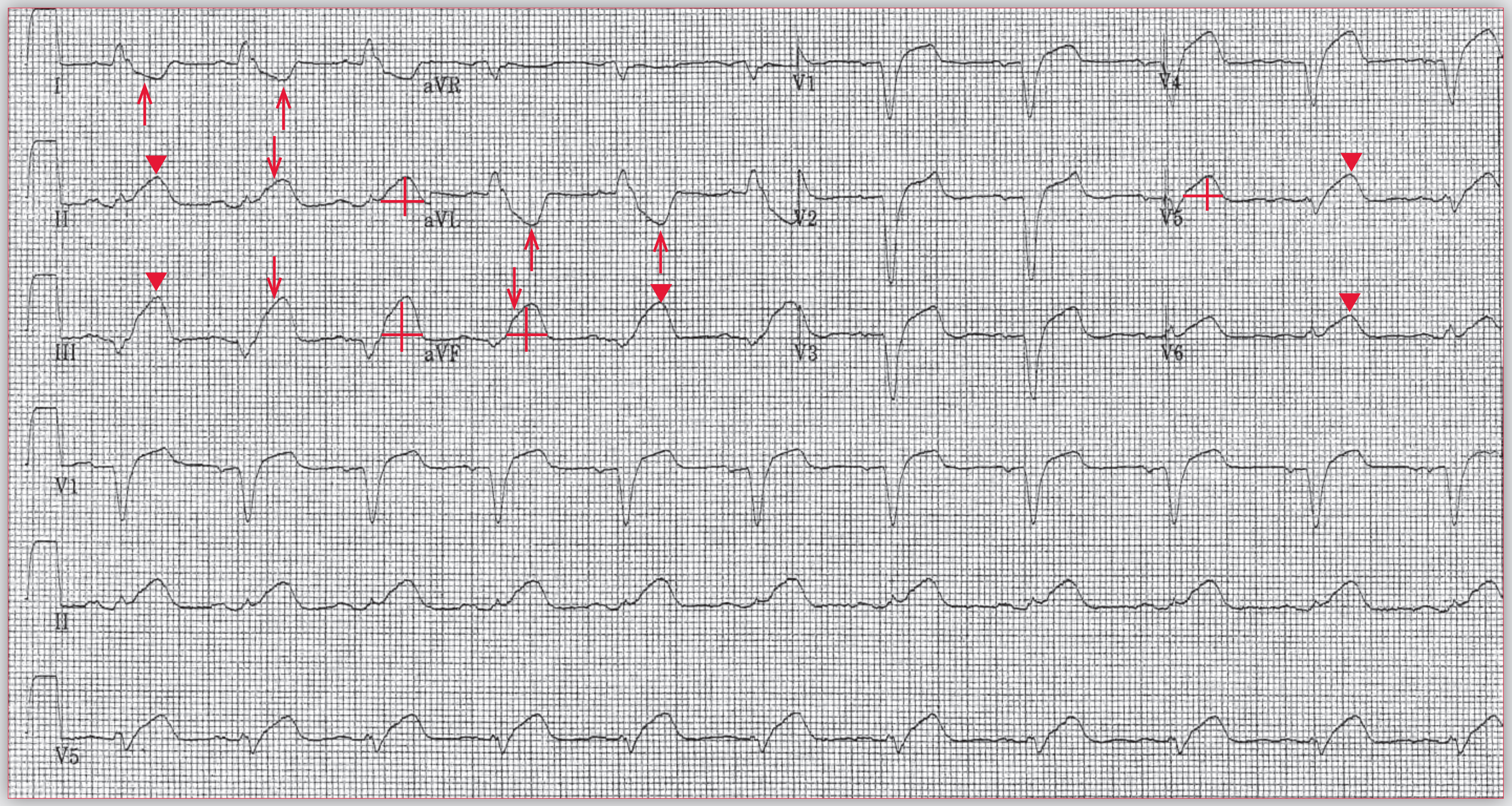


ECG 102B Analysis: Normal sinus rhythm, LBBB, acute inferior wall myocardial infarction

ECG 102B is from the same patient as ECG 102A and was obtained during the episode of chest discomfort. There is a regular rhythm at a rate of 66 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 msec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is increased (0.14 sec) and there is a LBBB morphology and left axis, as was present in ECG 102A. The QT/QTc intervals are normal (400/420 and 360/380 msec when corrected for the prolonged QRS complex duration). Notable are ST-segment elevations (↓) most prominent in leads II, III, and aVF and prominent J-point and ST-segment depressions in leads I and aVL (↑). In addition, the T waves are symmetric in these leads as well as in leads V5–V6 (▼). These are features characteristic of an acute inferior wall ST-segment elevation myocardial infarction (STEMI). With a LBBB, abnormalities of the left ventricle cannot be reliably diagnosed, as ventricular activation is not via the normal His-Purkinje system but is the result of direct myocardial activation. This includes an acute myocardial infarction, However, it has been reported by Sgarbossa that there are features that are consistent with an acute myocardial infarction when a LBBB or right ventricular paced complex is present. The ST segment elevations in the inferior leads are consistent with the Sgarbossa criteria:

1. ST-segment elevation ≥ 1 mm that is in the same direction (concordant) as the QRS complex in any lead.
2. ST-segment depression ≥ 1 mm in any lead from V1–V3.
3. ST-segment elevation ≥ 5 mm that is discordant with the QRS complex (*ie*, associated with a QS or rS complex).

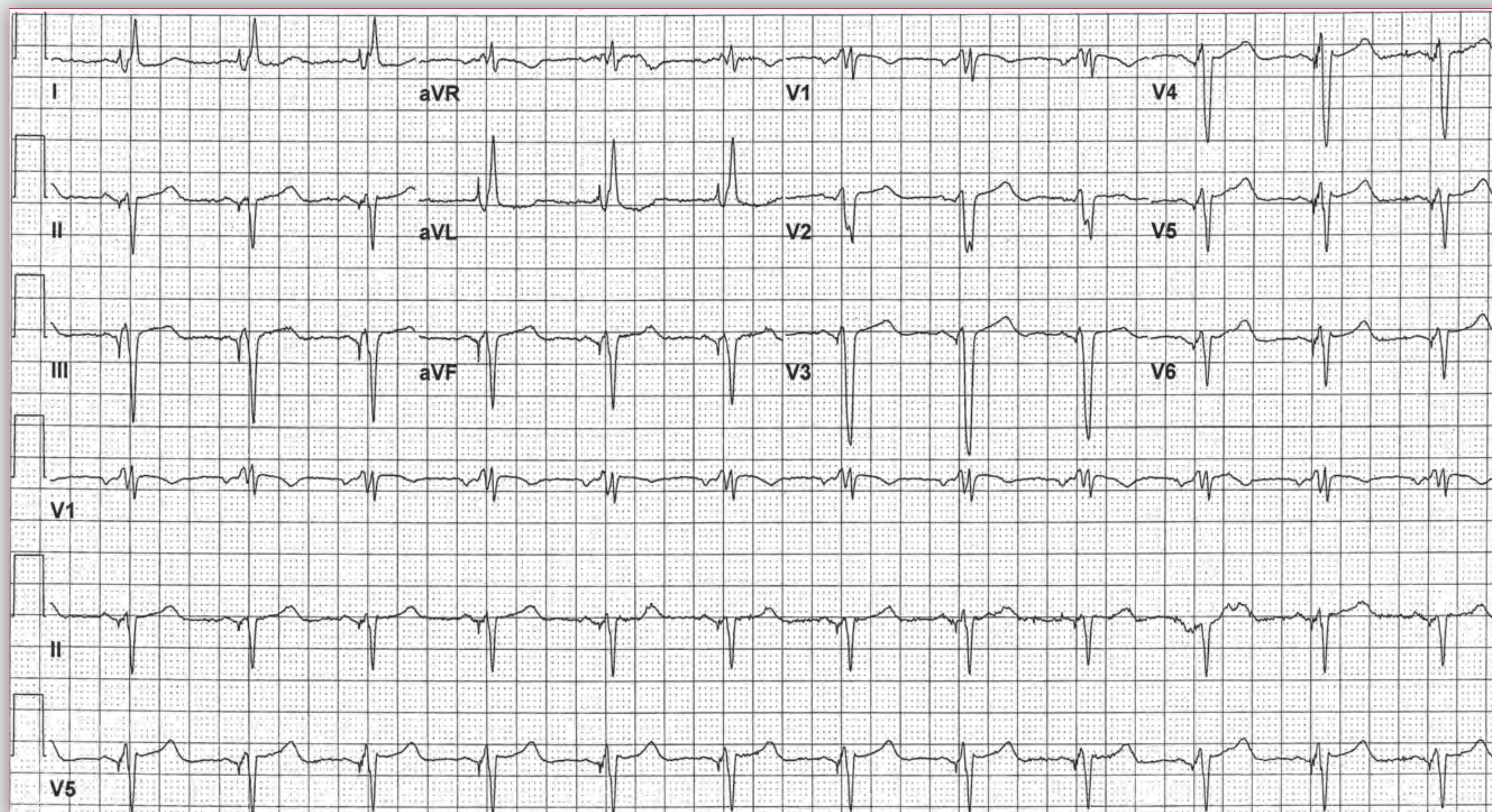
Further support for the presence of an acute inferior wall STEMI are the reciprocal changes of J-point and ST-segment depression in leads I and aVL as well as the symmetric T waves (hyperacute T waves), which are the result of local hyperkalemia due to increased membrane permeability that occurs with an infarction and the outward leak of potassium. This happens as membrane integrity is maintained by an energy-dependent sodium-potassium ATPase pump, which pumps in potassium and pumps out sodium. With absence of oxygen, ATP breaks down to adenosine, and membrane integrity is no longer maintained, accounting for the outward leak of potassium. With the absence of blood flow into the area, there is also a lack of blood flow out of the infarct zone, and hence the high levels of potassium remain in the infarcted area, with the development of local hyperkalemia, accounting for the T-wave abnormalities.

Given the clinical story and the ECG changes, a diagnosis of an acute STEMI can be made. Cardiac biomarkers will likely be normal, as the initial symptoms began only about 1 hour before presentation. Therefore, the patient should go for urgent catheterization and revascularization rather than wait to confirm the infarction with elevated biomarkers. ■

Core Case 103

An 86-year-old woman with a history of an ischemic cardiomyopathy is admitted to the hospital for decompensated heart failure. She had a biventricular pacemaker inserted 2 years ago, but has had multiple episodes of heart failure since that time. After admission, an ECG is obtained (ECG 103A). She is treated with a continuous infusion of furosemide and has a good response. However, 2 days after admission, she complains of severe substernal chest discomfort with radiation to her neck and jaw. She states that this is similar to her previous episodes of angina, although it is

ECG 103A

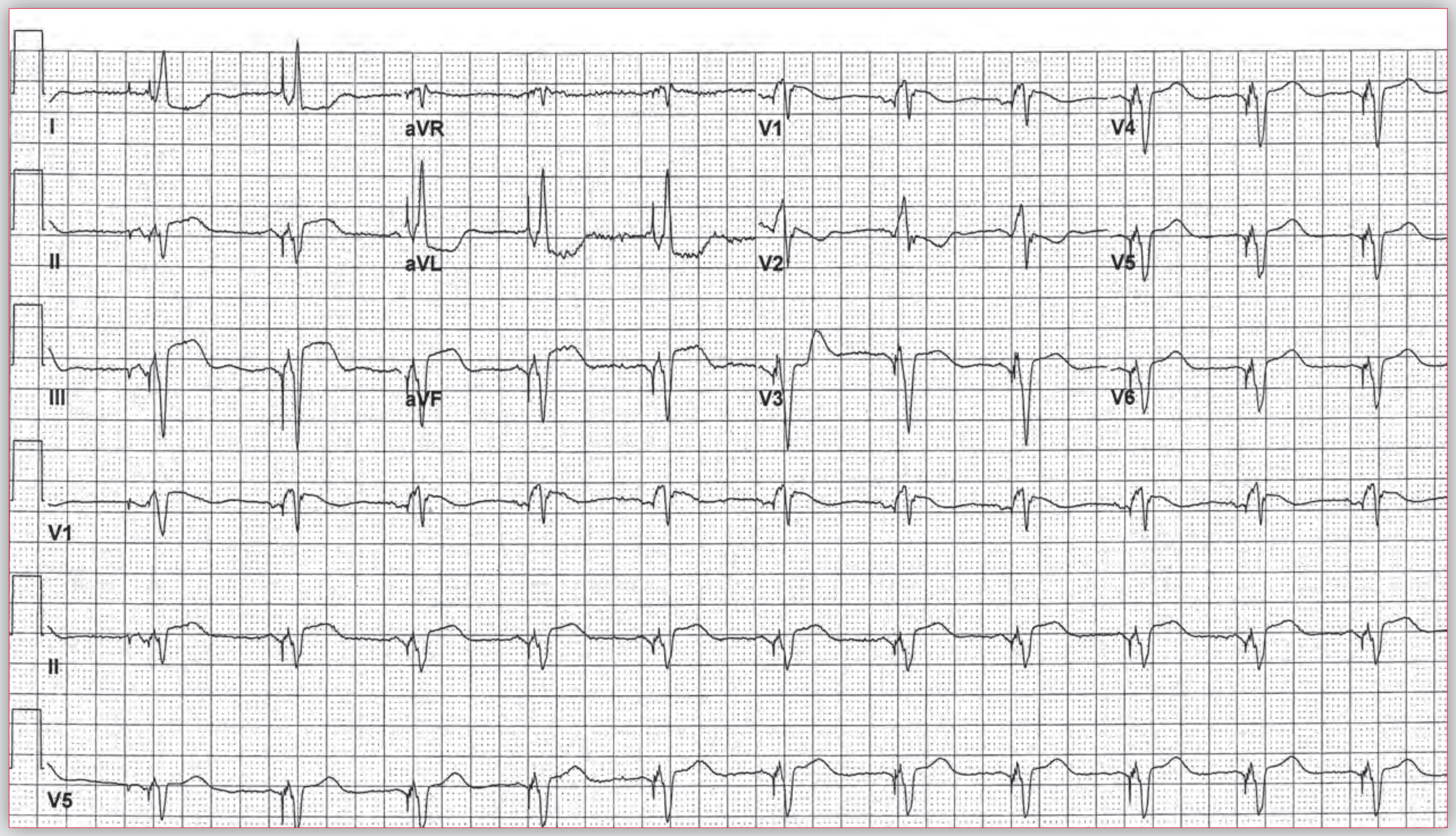


more severe and is associated with diaphoresis and nausea. An ECG is obtained (ECG 103B).

Although the ECG is obtained, it was not seen by the house staff. One the following day, chest pain had lessened, but her symptoms of heart failure worsened. Another ECG was obtained (ECG 103C).

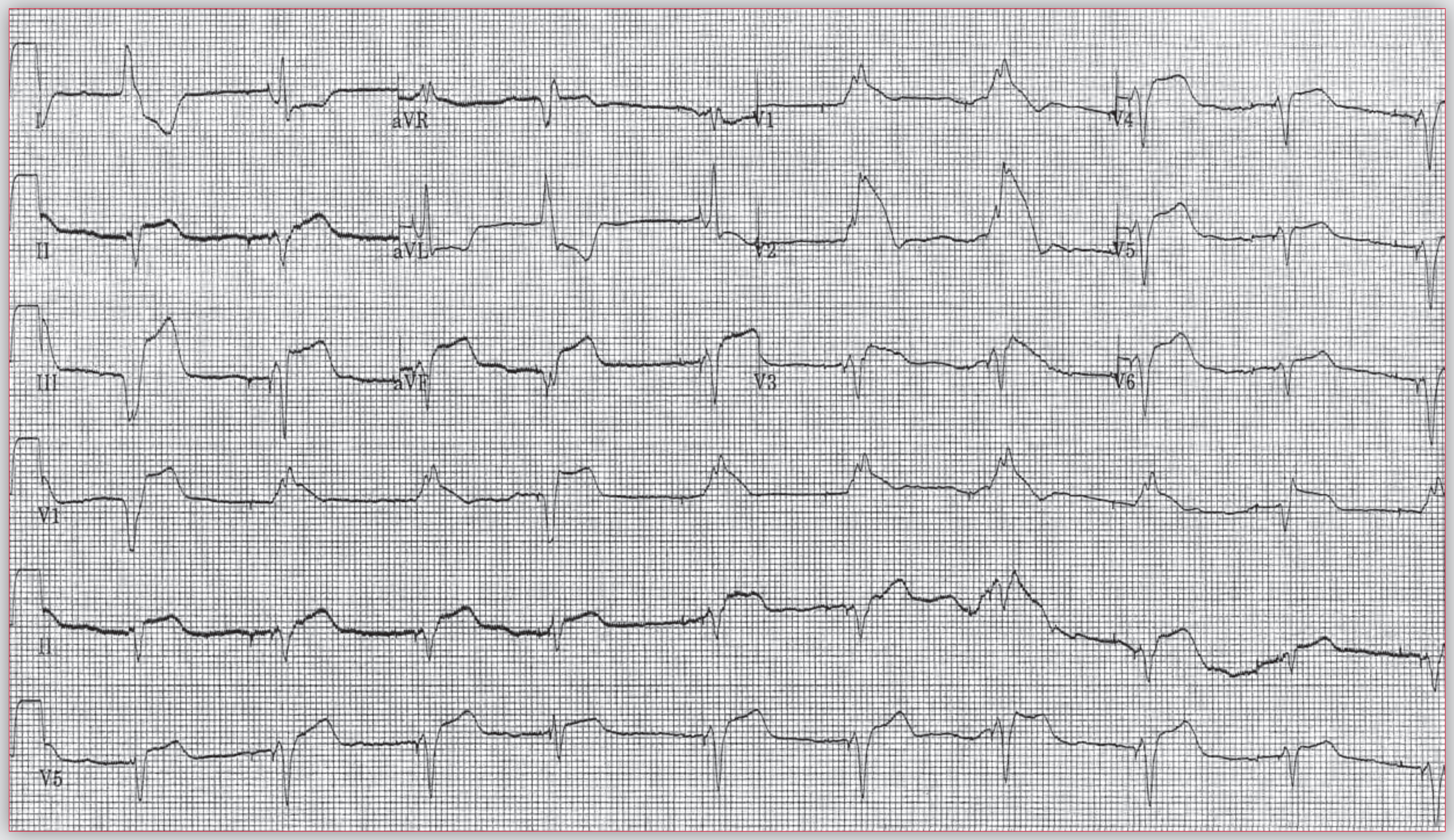
What does ECG 103B show?

ECG 103B

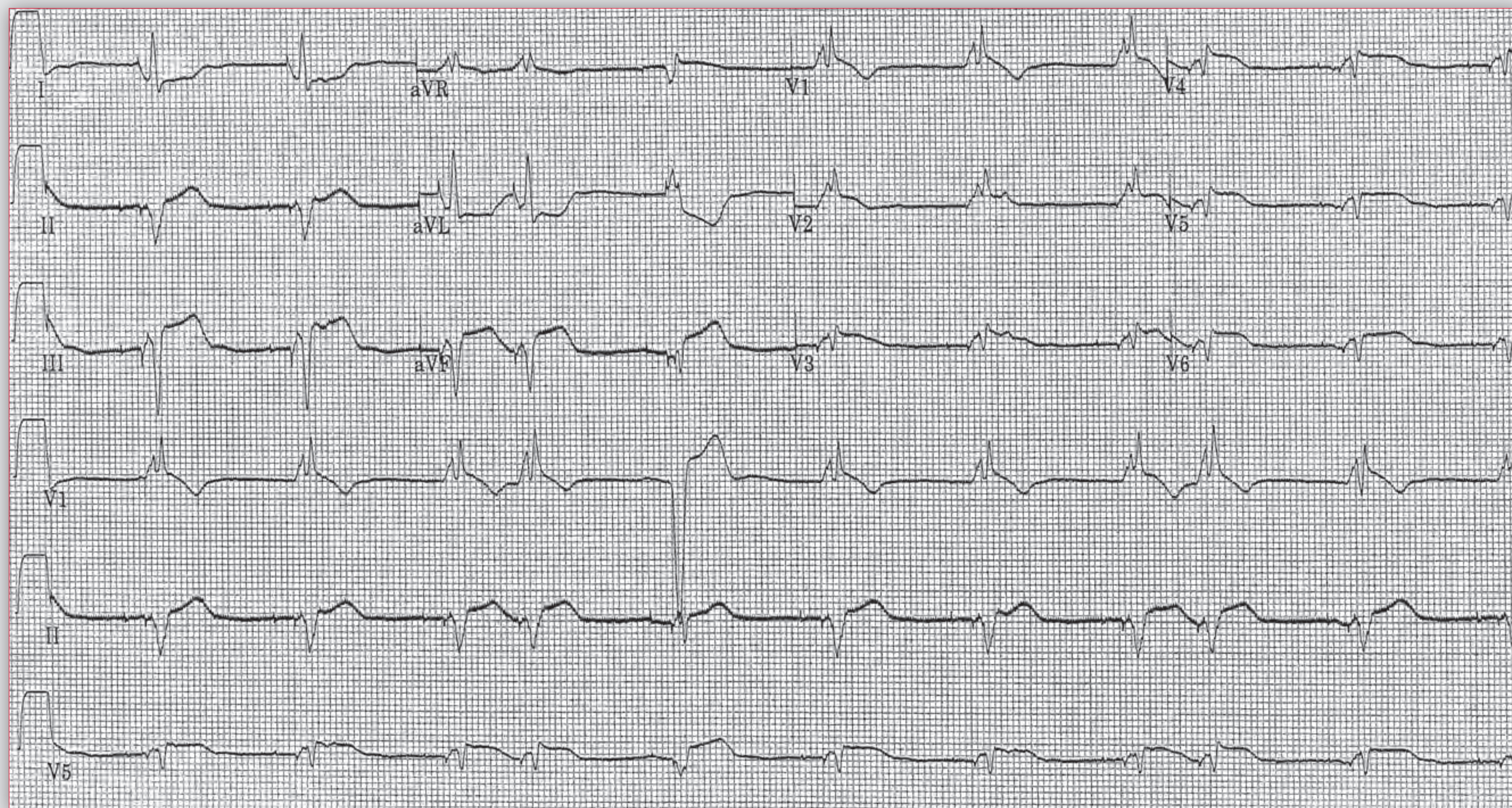


Core Case 103

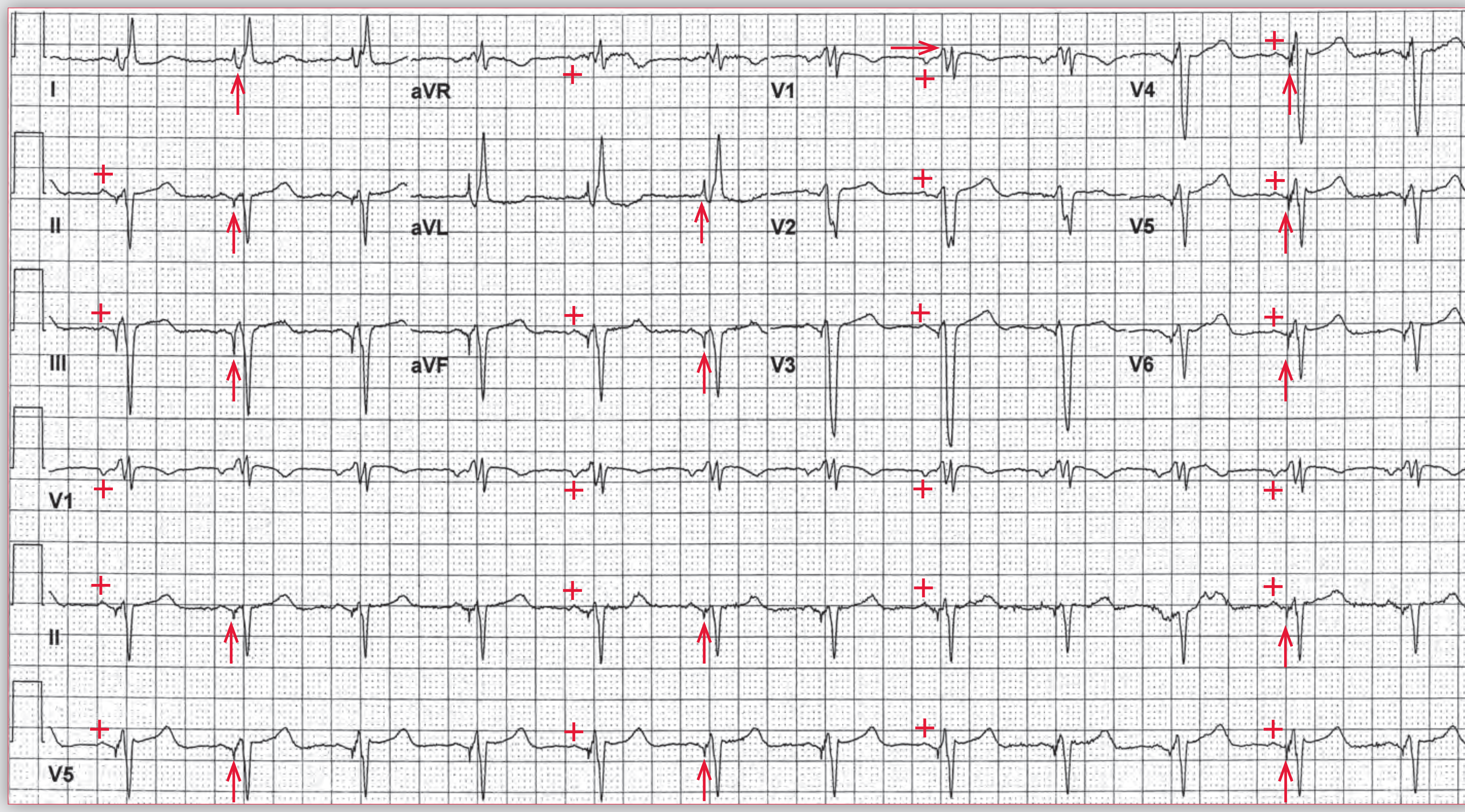
ECG 103C



ECG 103D



As a result of ECG 103C, another ECG was obtained (ECG 103D). What type of ECG is this and what does it show?



ECG 103A Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous pacing), biventricular pacemaker

ECG 103A is a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. Following the P wave, there is a pacemaker stimulus (↑) that is present before each QRS complex. Hence there is a dual-chamber pacemaker with atrial sensing and ventricular pacing (P-wave synchronous pacing). The QRS complex duration is prolonged (0.14 sec). The axis is extremely leftward between –30° and –90° (positive QRS complex in lead I and negative in leads II and aVF). The QT/QTc intervals are prolonged (440/480 msec) but are normal when the prolonged QRS complex duration is considered (400/440 msec). The morphology is, however, not typical for a right ventricular (RV) pacemaker, as there is an initial R wave in lead V1 (→) and an initial Q wave in lead I (↑). This pattern indicates that initial ventricular activation is occurring in a left-to-right direction, which is not typical for a RV pacemaker, in which initial activation is in a right-to-left direction. Hence this indicates that there is a left ventricular (LV) or biventricular pacemaker present. The most important lead indicating biventricular pacing is lead I as it is the only bipolar R-L lead. An impulse directed from right to left generates a positive QRS complex in lead I, while the presence of an initial Q wave or a QS complex in this lead indicates that forces are directed from left to right, *ie*, a LV or biventricular pacemaker.

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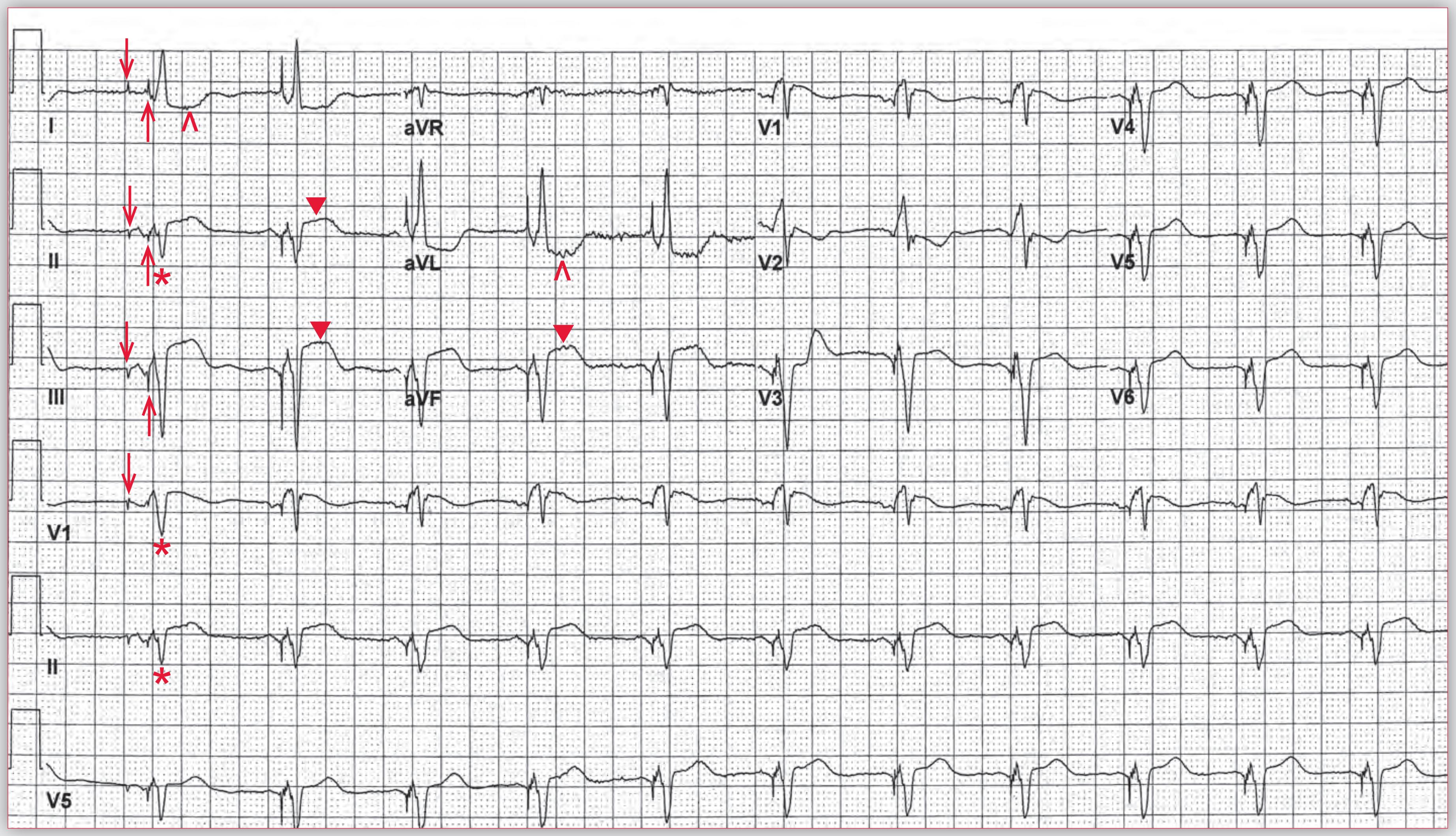

ECG 103B Analysis: Normal sinus rhythm, dual-chamber pacemaker, atrial sensed, ventricular paced (P-wave synchronous pacing) biventricular pacemaker, acute inferior wall myocardial infarction

ECG 103B shows there is a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex that is positive in leads I, II, aVF, and V4–V6. Following the P wave, there is a pacing stimulus (↑) with a constant AV delay or PR interval (0.16 sec). After the pacemaker stimulus, there is a QRS complex that has the same morphology and axis as seen in ECG 103A. The QT/QTc intervals are the same. Hence this is biventricular pacing and the pacing mode is P-wave synchronous ventricular pacing. The first complex seen (*) also has a pacemaker stimulus before the P wave (↓); hence this is AV sequential pacing.

When compared to ECG 103A, it can be seen that there is ST-segment elevation in leads II, III, and aVF (▼). Although this is a biventricular-paced rhythm, the Sgarbossa criteria (originally reported to be useful with a left bundle branch block (LBBB) or RV paced rhythm) can probably be used in this situation, as well as in other situations in which there is direct myocardial activation. The Sgarbossa criteria for an acute myocardial infarction in these situations include ≥ 1 mm ST elevation concordant with the QRS complex (*ie*, a positive QRS complex), ≥ 5 mm discordant with the QRS complex (*ie*, a negative QRS complex), or ≥ 1 mm ST depression in leads V1–V3. As there is ≥ 5 mm ST-segment elevation in lead III and significant ST elevation in leads II and aVF (albeit less than 5 mm), this ECG represents an acute inferior wall myocardial infarction in the presence of biventricular pacing. In addition, there is new ST depression in leads I and aVL (^), which are reciprocal changes associated with the acute inferior wall myocardial infarction.

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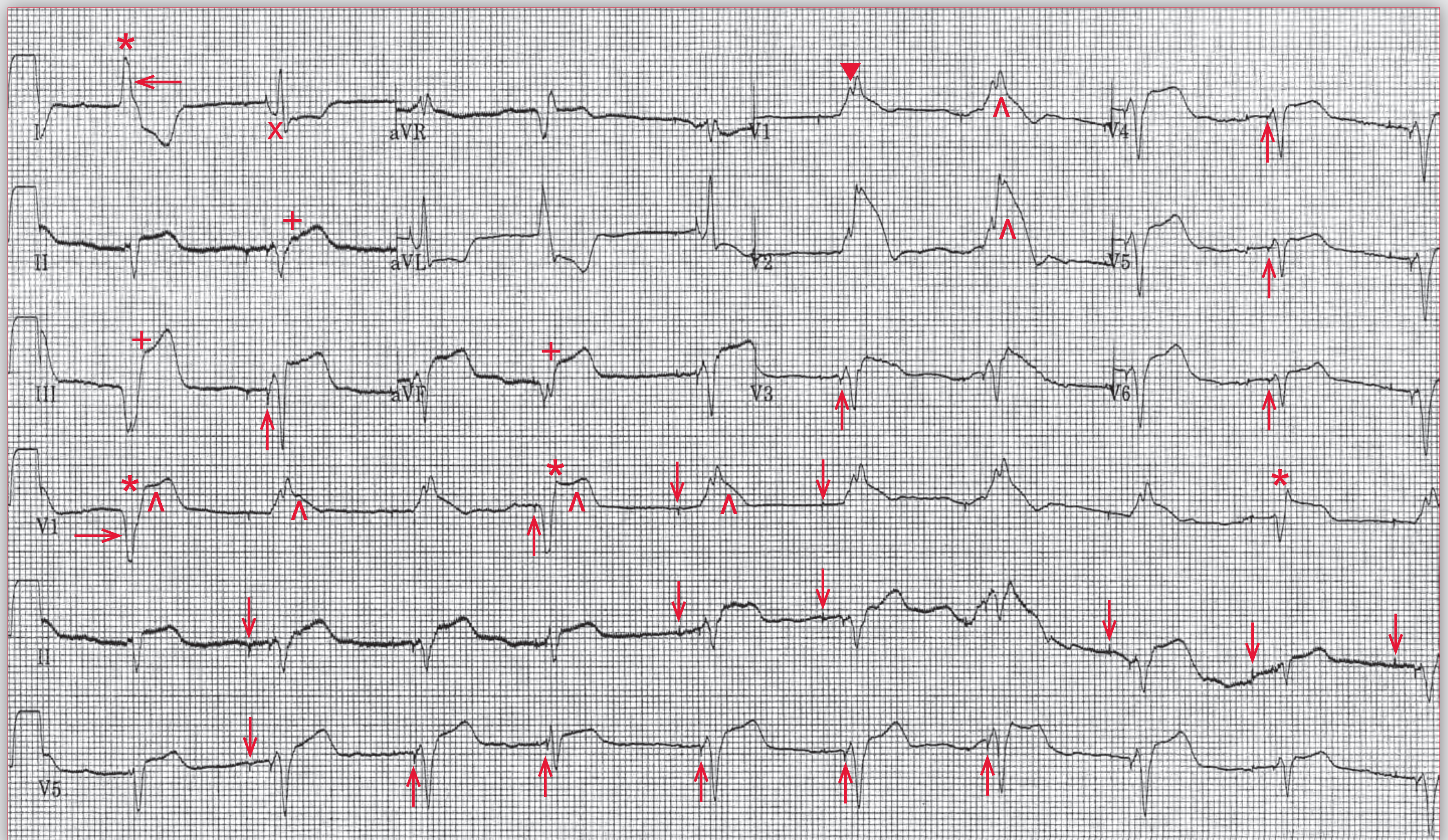

ECG 103C Analysis: Probable atrial fibrillation, AV sequential pacing, failure of atrial capture, intermittent failure of left ventricular lead capture, acute inferior wall myocardial infarction, acute right ventricular infarction

ECG 103C shows the rhythm is regular at a rate of 64 bpm, but there are no obvious P waves seen. There are, however, atrial stimuli seen (↓), but no associated P waves, suggesting that there is no atrial capture. It is possible that the underlying rhythm is atrial fibrillation. There is a pacemaker stimulus (↑) seen before each QRS complex. However, the QRS complex morphology is variable. The first QRS complex (*) has a broad R wave in lead I (←) and a QS complex in lead V1 (→), consistent with right ventricular pacing. The fourth and ninth QRS complexes (*) (as noted in the lead V1 rhythm strip) have the same morphology. In contrast, the second, third, and fifth through eighth QRS complexes have an initial Q wave in lead I (x) and a tall (although abnormal) R wave in lead V1 (▼). This morphology is typical for LV or biventricular pacing. Therefore, it appears that there is intermittent failure of LV lead capture, resulting in a QRS complex that has a RV paced morphology.

ST-segment elevation is still apparent in leads II, III, and aVF (+) and the ST segments are even more elevated than in ECG 103B. In addition, there is now significant ST elevation in leads V1–V2 (^) (seen with both the right ventricular and the biventricular paced complexes). These ST-segment elevations, in association with an acute inferior wall myocardial infarction, indicate right ventricular involvement, *ie*, an infarction of the free wall of the right ventricle. The ST-segment changes now fulfill the Sgarbossa criteria for myocardial infarction, which were reported useful for a LBBB and a RV paced rhythm (which has the same morphology as a LBBB) can probably be applied to a biventricular paced QRS complex as in all of these situations there is direct myocardial activation, bypassing the normal His-Purkinje system:

1. ST-segment elevation ≥ 1 mm that is in the same direction (concordant) with the QRS complex in any lead.
2. ST-segment depression ≥ 1 mm in any lead from V1–V3.
3. ST-segment elevation ≥ 5 mm that is discordant with the QRS complex (*ie*, associated with a QS or rS complex).

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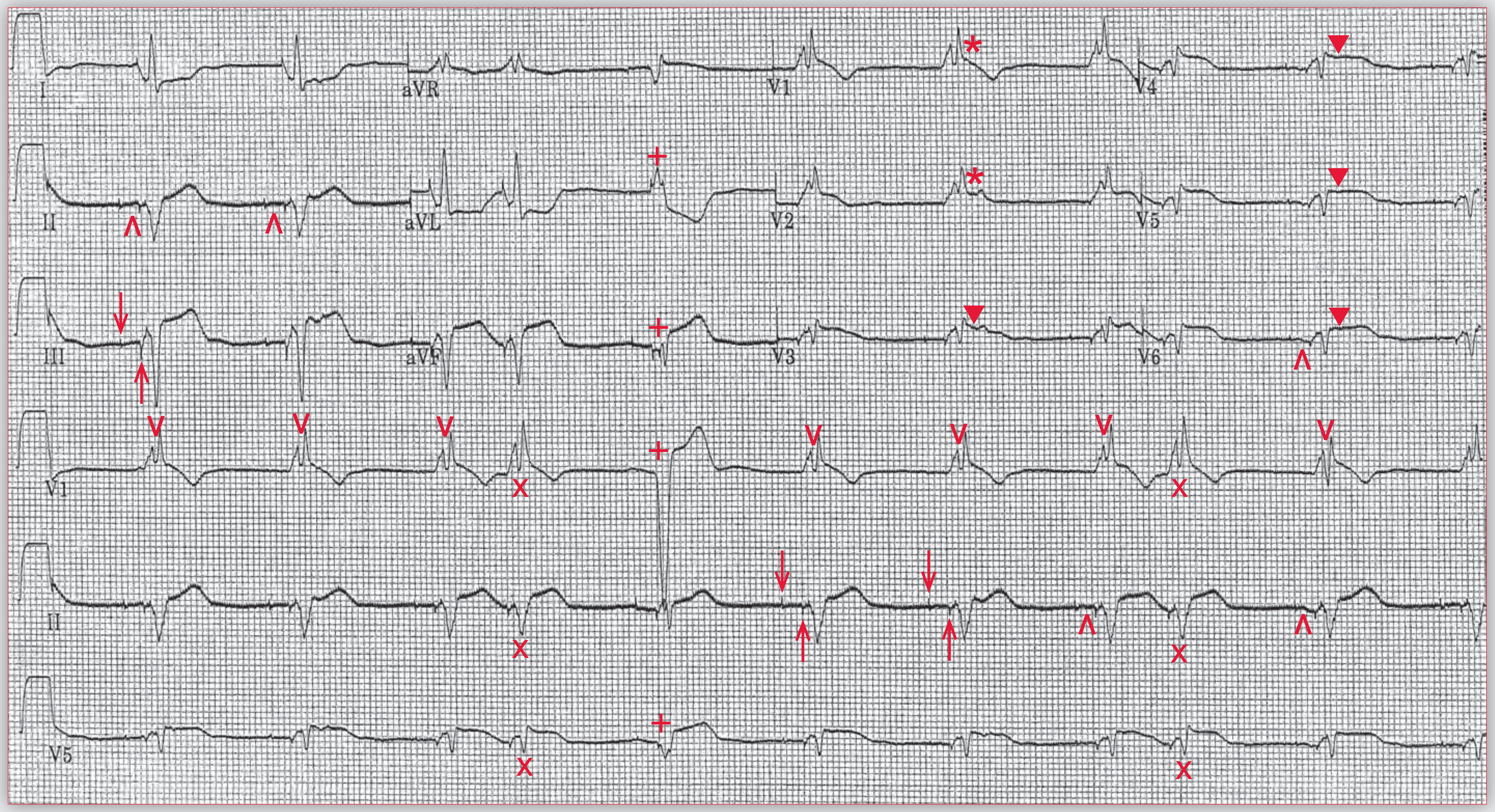


ECG 103D Analysis: Right-sided leads, AV sequential pacing, premature atrial complexes, acute inferior wall myocardial infarction, acute RV infarction, intermittent failure of LV lead capture

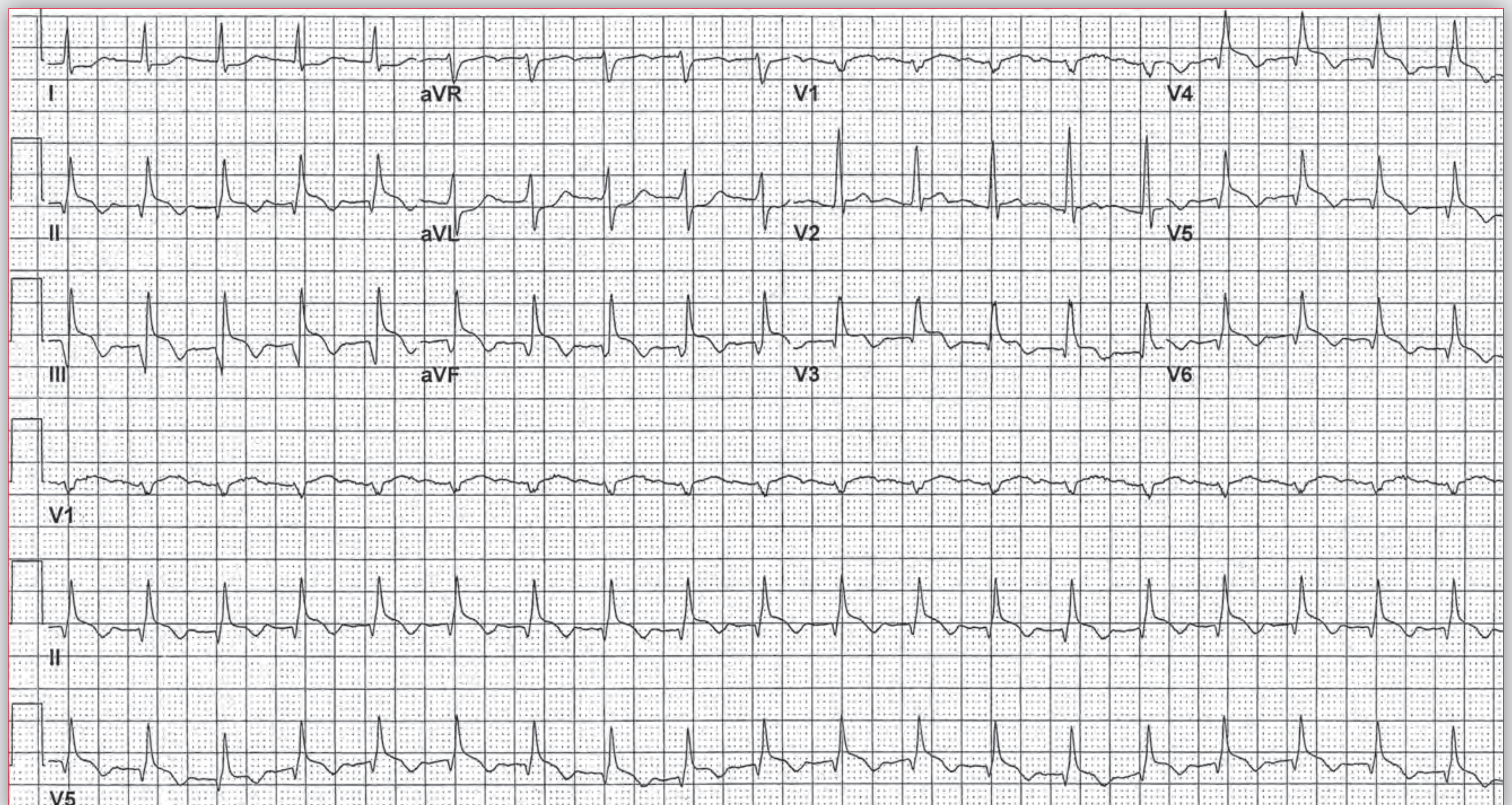
ECG 103D was recorded using right-sided leads. There is a regular rhythm at a rate of 60 bpm. There are atrial (↓) and ventricular (↑) pacing stimuli seen. In addition, there is a P wave that follows the atrial stimulus (^). It can be noted that there is still intermittent failure of the LV lead to capture, *ie*, complex 5 (+) (in the V1 rhythm strip) has a RV paced morphology. Most of the complexes, however, do have a morphology consistent with biventricular pacing (v).

It can be noted that there is ST-segment elevation in the right-sided V3–V6 (▼), which is characteristic of RV infarction, confirming the diagnosis based on the ST elevation in leads V1–V2 noted on ECG 103C. In addition, the changes in leads V1–V2 are still present, *ie*, ST-segment elevation (*). Also noted are two premature biventricular paced QRS complexes (x) that only have a ventricular stimulus. These are premature atrial complexes. ■

Core Case 104

A 59-year-old man presents to the emergency department with a complaints of severe substernal chest pressure radiating to his left arm and associated with diaphoresis. The chest pressure had been present for 4 hours, and he finally decided to come to the hospital. Upon

ECG 104A

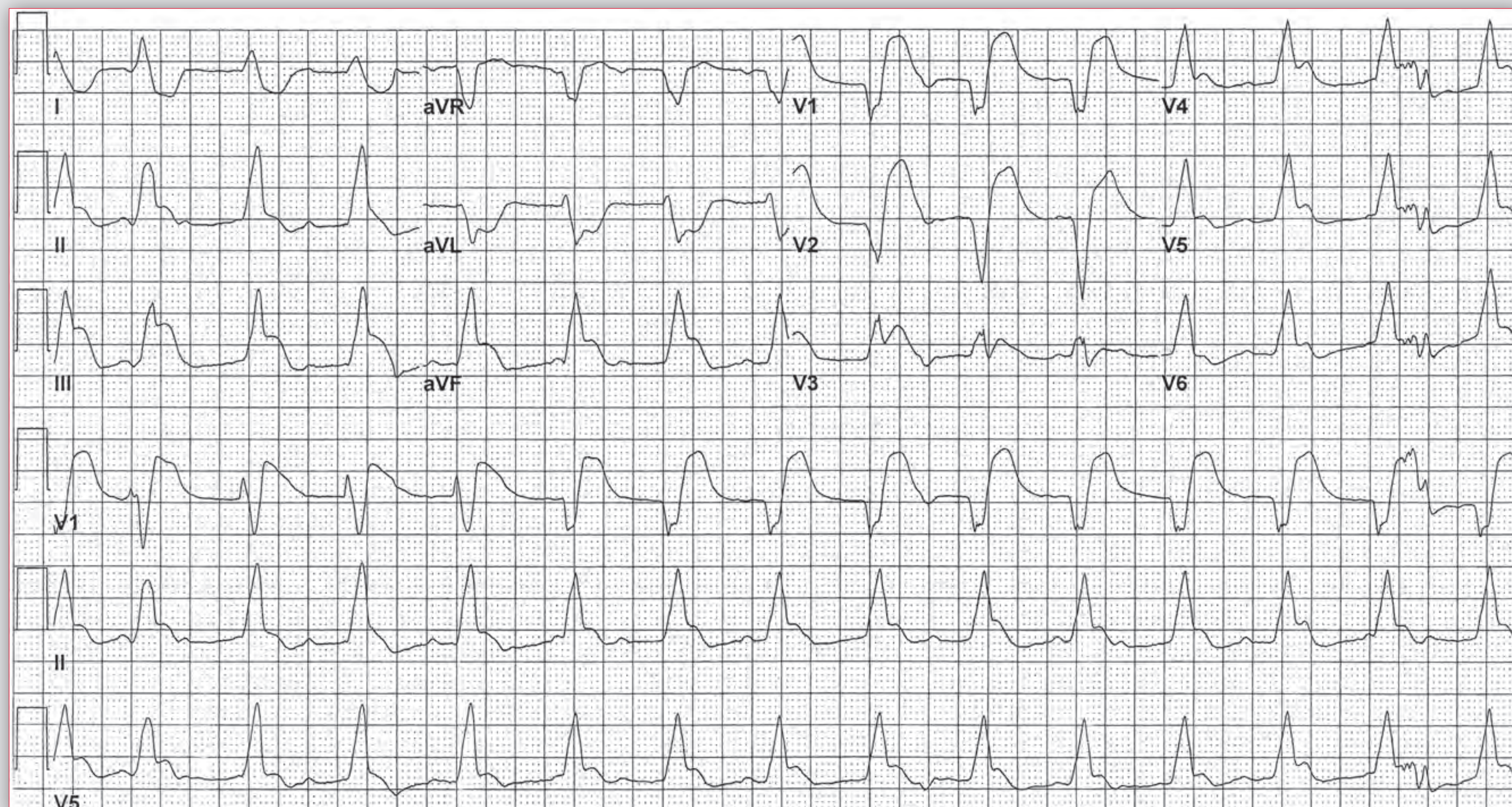


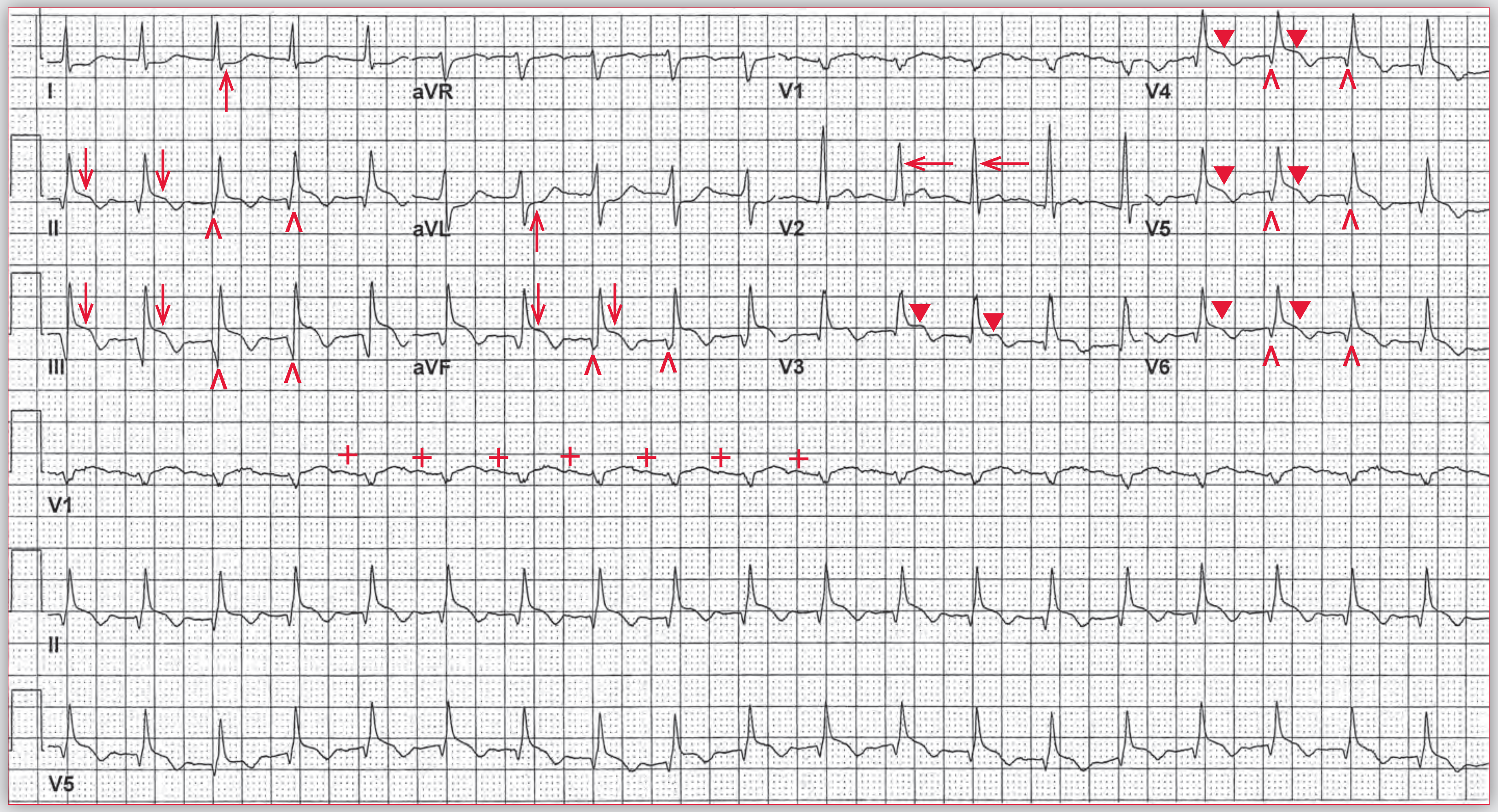
presentation, his heart rate is 110 bpm and blood pressure is 110/80. Scattered bibasilar rales are heard. An ECG is obtained (ECG 104A). The patient is begun on heparin and intravenous nitroglycerin. Several minutes later, another ECG is obtained (ECG 104B).

What does ECG 104A show?

What changes are noted on ECG 104B?

ECG 104B





ECG 104A Analysis: Supraventricular tachycardia (sinus vs. atrial), early transition, acute inferior and anterolateral myocardial infarction

ECG 104A shows a regular rhythm at a rate of 120 bpm. Although P waves are not obvious in most leads, there is a P wave before each QRS complex apparent in lead V1 (+). There is a stable PR interval (0.18 sec). As P waves are not seen in other leads, it is not clear if this is a sinus or an atrial rhythm. The QRS complex duration is normal (0.10 sec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). There is a tall R wave in lead V2 (←) that is early transition or counterclockwise rotation. This is established by imagining the heart as viewed from under the diaphragm. When there is counterclockwise rotation, left ventricular forces develop earlier and are seen in the right precordial leads, *ie*, lead V2.

There is ST-segment elevation seen in leads II, III, and aVF (↓), consistent with an acute inferior wall myocardial infarction. There are also ST-segment elevations seen in leads V3–V6 (▼) consistent with an acute anterolateral myocardial infarction. Associated with the inferior ST elevation are reciprocal ST-segment depressions in leads I and aVL (↑). In addition, Q waves are seen in leads II, III, aVF, and V4–V6 (^). The QT/QTc intervals are normal (320/450 msec).

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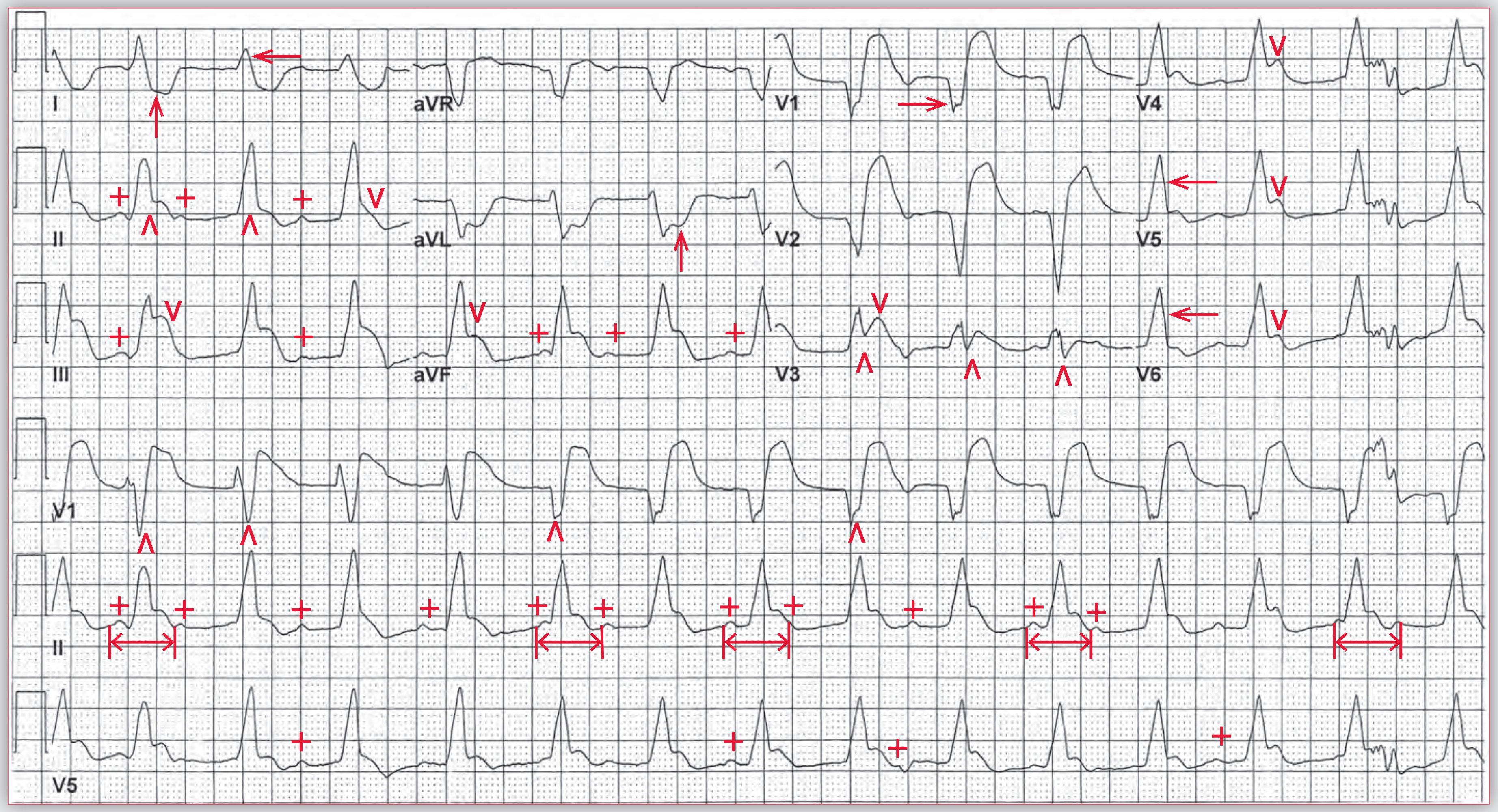


ECG 104B Analysis: Sinus tachycardia, complete heart block, escape ventricular rhythm, acute inferior and anterolateral myocardial infarction

ECG 104B is from the same patient as ECG 104A. There is a regular rhythm with a rate of 86 bpm. P waves are seen in leads II, III, aVF, and V5 (+). There is a regular PP interval with an atrial rate of 140 bpm (↔). Although P waves are not always seen, when present, they are on time. The P waves are positive in leads I, II, aVF and V4–V6. Hence this is sinus tachycardia. The PR interval is not constant and hence there is AV dissociation present. As the atrial rate is faster than the ventricular rate, this is complete heart block. The QRS complex duration is increased (0.18 sec) and is wider than the QRS complexes in ECG 104A. There is also a change in morphology, with a tall R wave in leads I and V5–V6 (←) and a broad QS complex in lead V1 (→). Although this resembles a left bundle branch block (LBBB), it can also be noted that there are marked changes in QRS complex morphology (^), particularly evident in leads II, V1, and V3. Therefore, these are ventricular complexes, *ie*, an escape ventricular rhythm. Although this is a ventricular rhythm, ST-segment elevation is still seen, and it is apparent in the same leads as was seen in ECG 104A, *ie*, leads II, III, aVF, and V3–V6 (v). There is also significant ST-segment depression in leads I and aVL (↑), which is also more apparent than seen in ECG 104A.

The diagnosis of an acute myocardial infarction is particularly difficult whenever left ventricular (LV) activation does not occur via the normal His-Purkinje system, *ie*, LBBB, ventricular pacing or a ventricular complex. In these situations, LV activation occurs through an abnormal pathway, *ie*, directly through the LV myocardium. Since ventricular activation is abnormal, the diagnosis of abnormalities affecting the LV is difficult to establish on the ECG. However, Sgarbossa reported criteria that have been be useful:

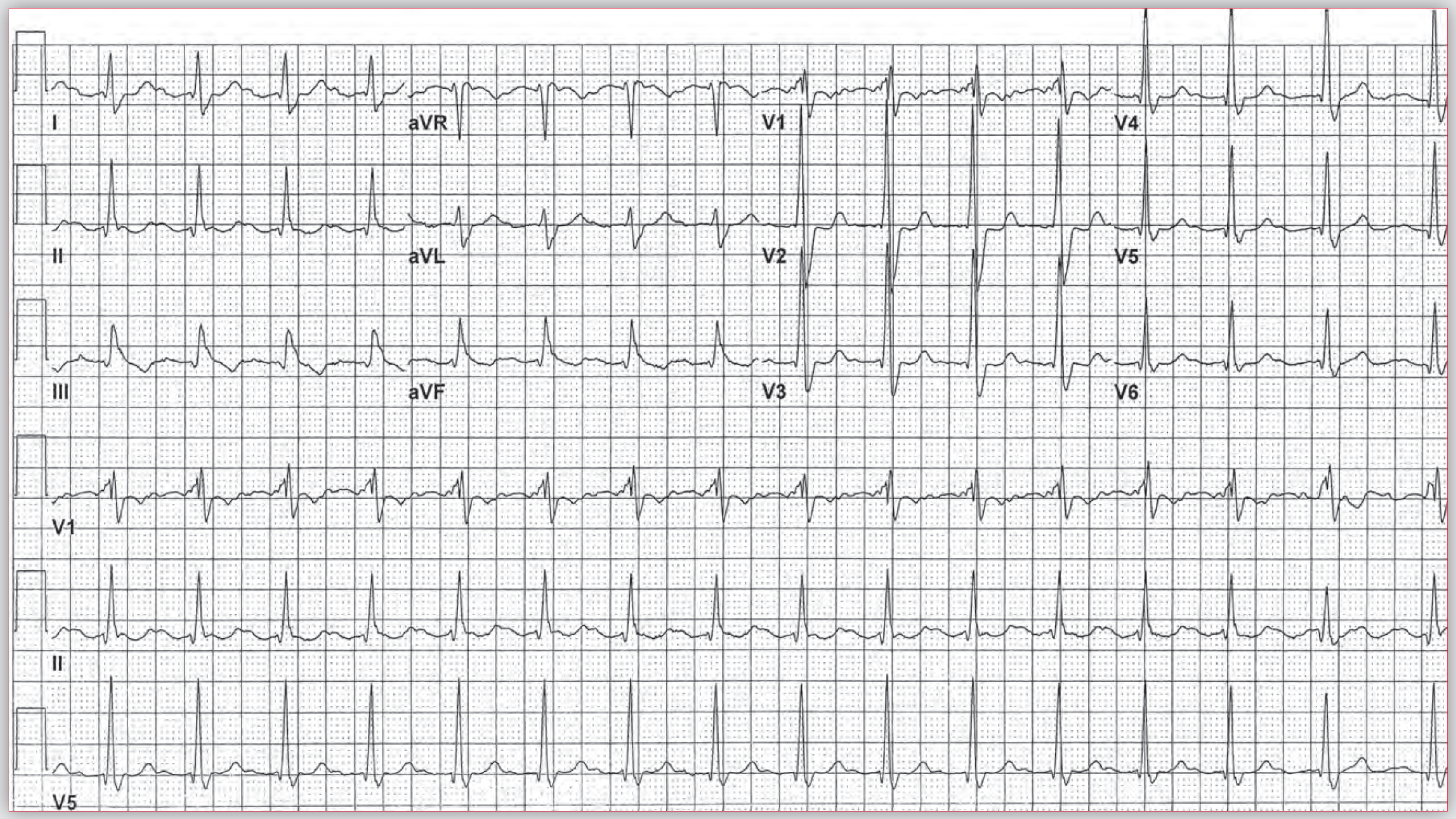
1. ST-segment elevation of ≥ 1 mm that is in the same direction (concordant) as the QRS complex in any lead.
2. ST-segment depression of ≥ 1 mm in any lead from V1–V3.
3. ST-segment elevation of ≥ 5 mm that is discordant with the QRS complex (*ie*, associated with a QS or rS complex).

Although the Sgarbossa criteria were reported to be useful in a LBBB and a right ventricular paced rhythm (in which there is also a LBBB pattern), they are probably also applicable to a ventricular rhythm as the same issues are involved, *ie*, LV activation that is not via the normal His-Purkinje system. Therefore, it can be seen that the ST-segment changes of an acute myocardial infarction that are seen during the ventricular rhythm are diagnostic of an acute myocardial infarction. In addition, they are similar to the ST-segment changes seen during sinus rhythm. ■

Core Case 105

A 72-year-old woman with a history of deep vein thrombosis status postimplantation of an inferior vena cava (IVC) filter is scheduled for multiple teeth extractions. In preparation, her warfarin is held for several days. The day prior to the planned procedure, she experienced sudden onset of chest pain, palpitations, and dyspnea. She presents to a local emergency department. An ECG is obtained (ECG 105B) and compared to her baseline ECG (ECG 105A).

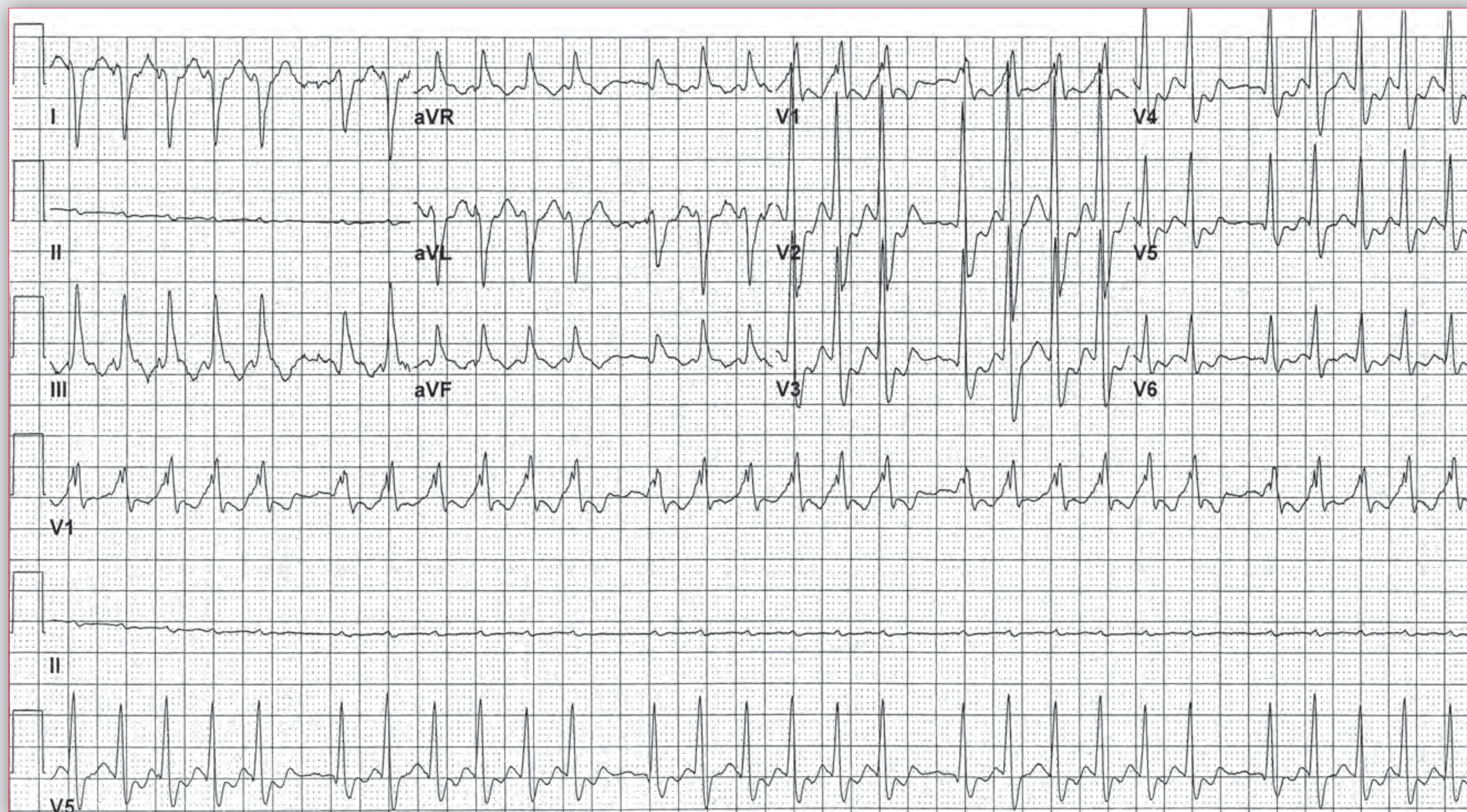
ECG 105A

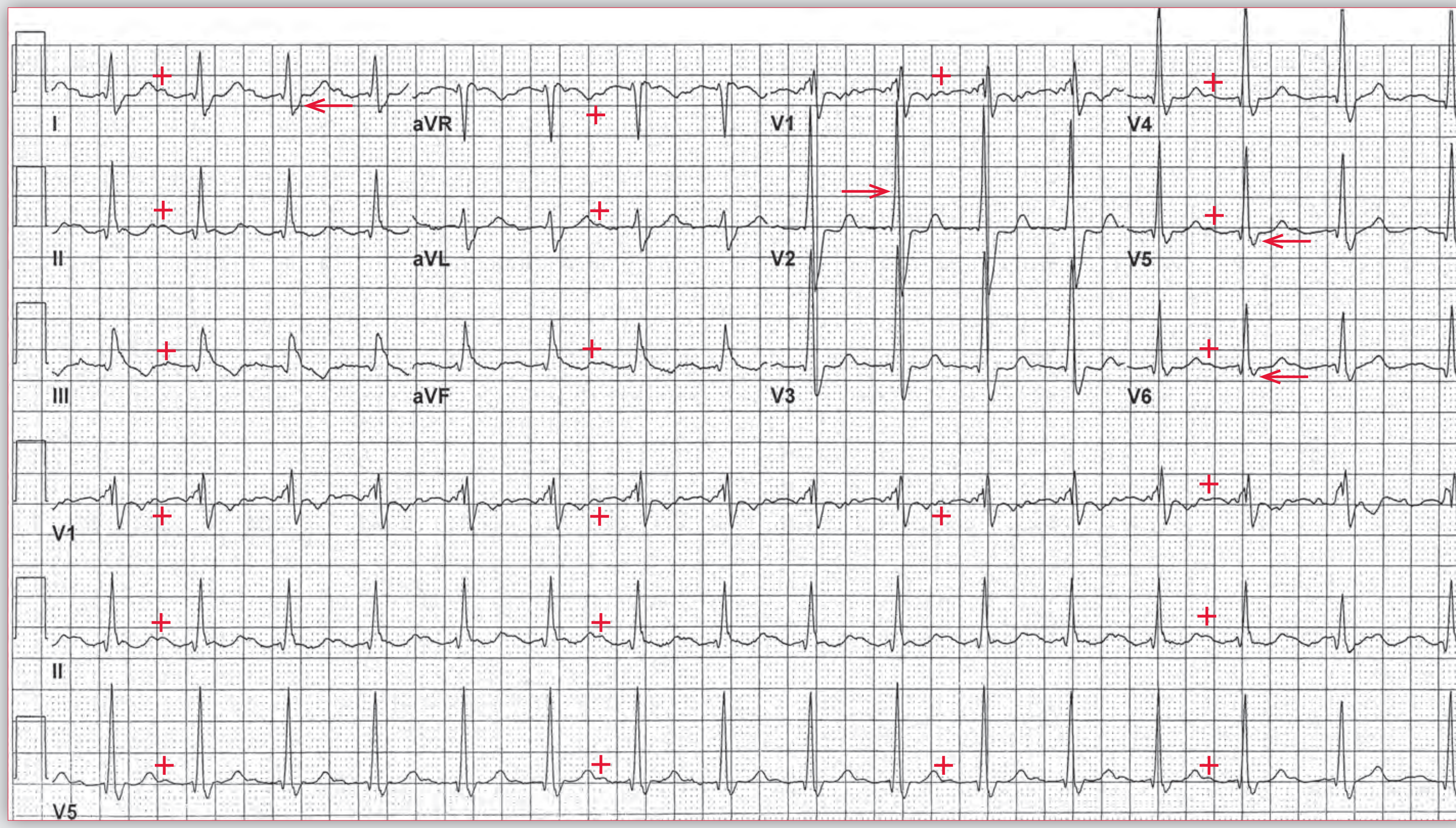


What are the changes noted on the current ECG when compared to the baseline ECG?

Does the ECG suggest a diagnosis that accounts for the symptoms?

ECG 105B



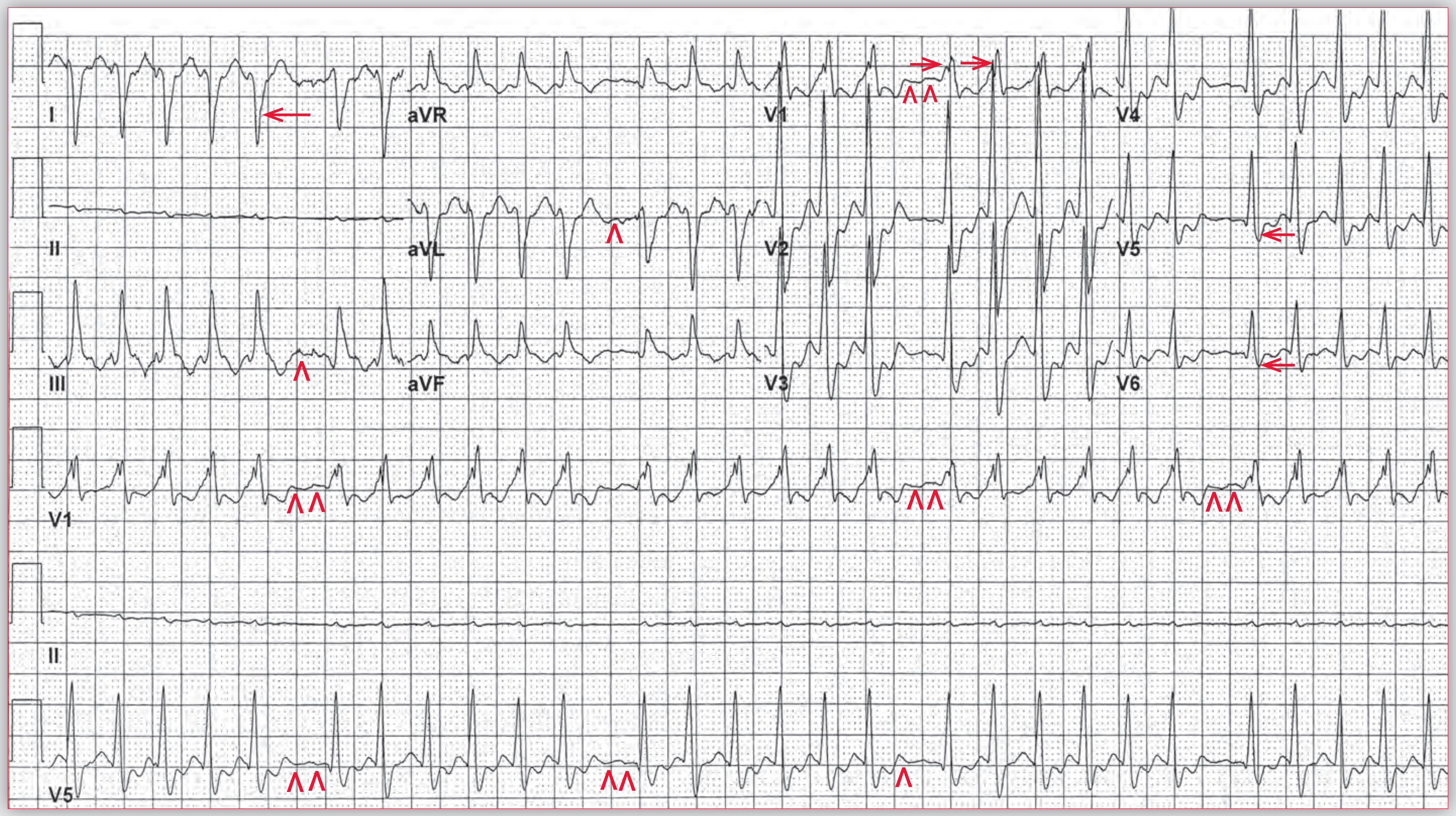


ECG 105A Analysis: Sinus rhythm, counterclockwise rotation, prolonged PR interval, intraventricular conduction delay (IVCD)

ECG 105A shows there is a regular rhythm with a rate of 96 bpm. There is a P wave (+) before each QRS complex, and the PR interval is constant (0.28 sec). The P waves are positive in leads I, II, aVF, and V5–V6. Hence this is a sinus rhythm with a first-degree AV block.

The QRS complex duration is prolonged (0.14 sec) and the axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QRS morphology resembles a right bundle branch block (RBBB), with broad S waves in leads I and V5–V6 (←). However, the morphology in lead V1 is not typical for a RBBB as there is an RS complex. This is an intraventricular conduction delay. There is early transition, with a tall R wave in lead V2 (→). This is due to counterclockwise rotation of the electrical axis in the horizontal plane, established by imagining the heart as seen from under the diaphragm. With counterclockwise rotation, left ventricular forces are seen earlier in the precordial leads, accounting for the tall R wave in lead V2. The QT/QTc intervals are prolonged (380/480 msec) but are normal when the prolonged QRS complex duration is considered (340/430 msec).

continues



ECG 105B Analysis: Atrial fibrillation, RBBB, rightward axis, left posterior fascicular block

ECG 105B shows the rhythm is an irregularly irregular rhythm at a rate of 168 bpm. There are no obvious P waves seen before or after the QRS complexes. However, there are irregular undulations of the baseline (^). Hence this is atrial fibrillation with a rapid ventricular response.

Compared to the baseline ECG (ECG 105A), the QRS complexes duration is wide (0.16 sec) and there is now a typical RBBB pattern with a tall, broad R wave in V1 (→) and broad S waves in leads I and V4–V6 (←). In addition, there has been a marked axis shift, which is now rightward between +90° and +180° (QRS negative in lead I and positive in lead aVF). The complex is negative in lead I even when the terminal S wave from the RBBB is considered. The QT/QTc intervals are prolonged (320/535 msec) but are normal when the prolonged QRS complex is considered (260/420 msec). The clinical history as well as the significant changes on the ECG, *ie*, the development of a RBBB and a marked rightward shift in the axis, are suggestive of acute pressure overload of the right ventricle, consistent with an acute pulmonary embolism. This was confirmed by CTA that demonstrated a large pulmonary embolism in the right pulmonary artery.

The changes on the ECG that may be seen in pulmonary embolism are:

1. Sinus tachycardia
2. Evidence of right-sided pressure overload with a rightward axis shift (often a pattern of deep S in lead I; Q- and T-wave inversion in lead III) and occasionally a new RBBB.
3. Nonspecific ST-T wave changes
4. Atrial arrhythmias ■

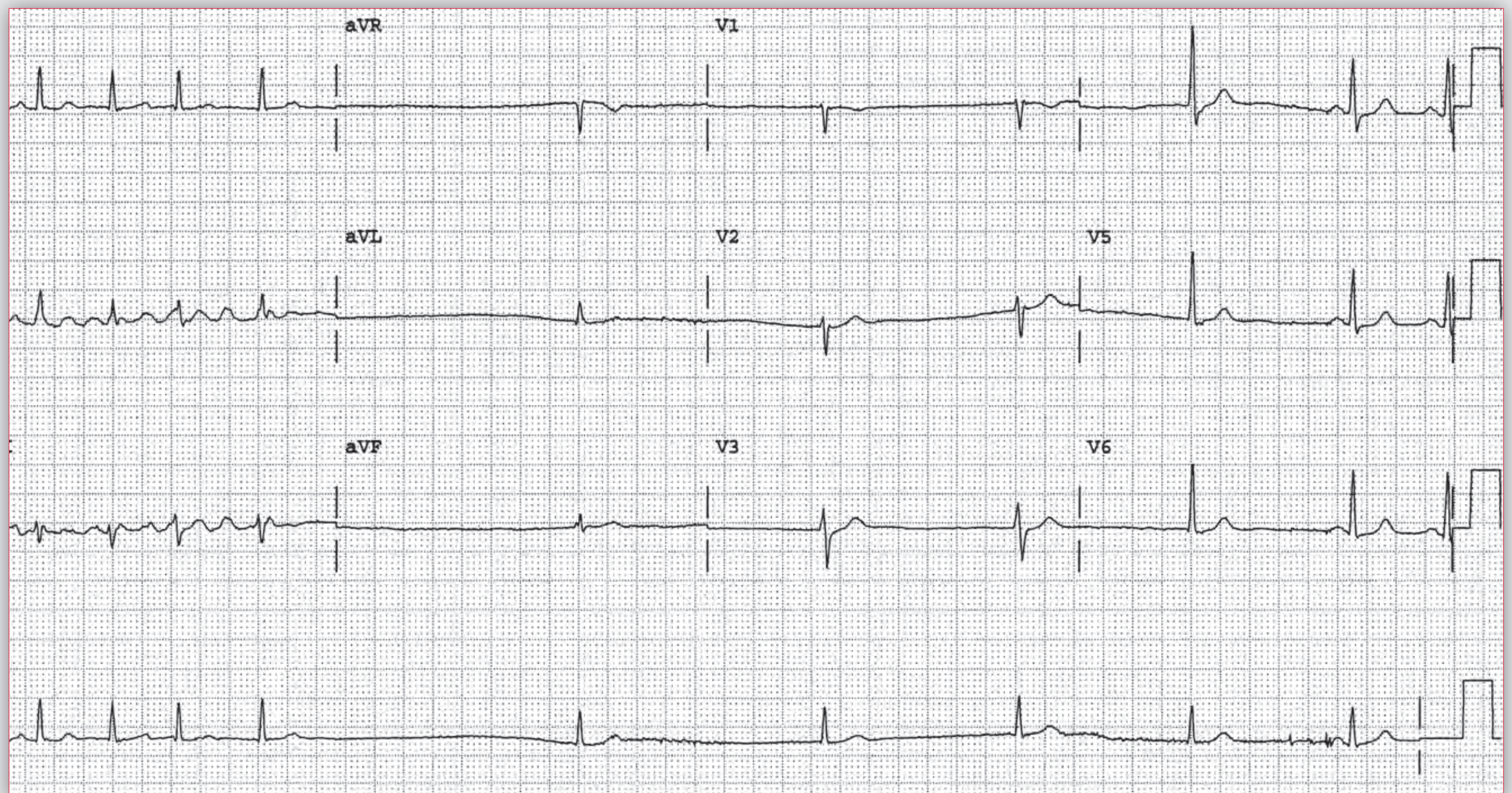
Notes

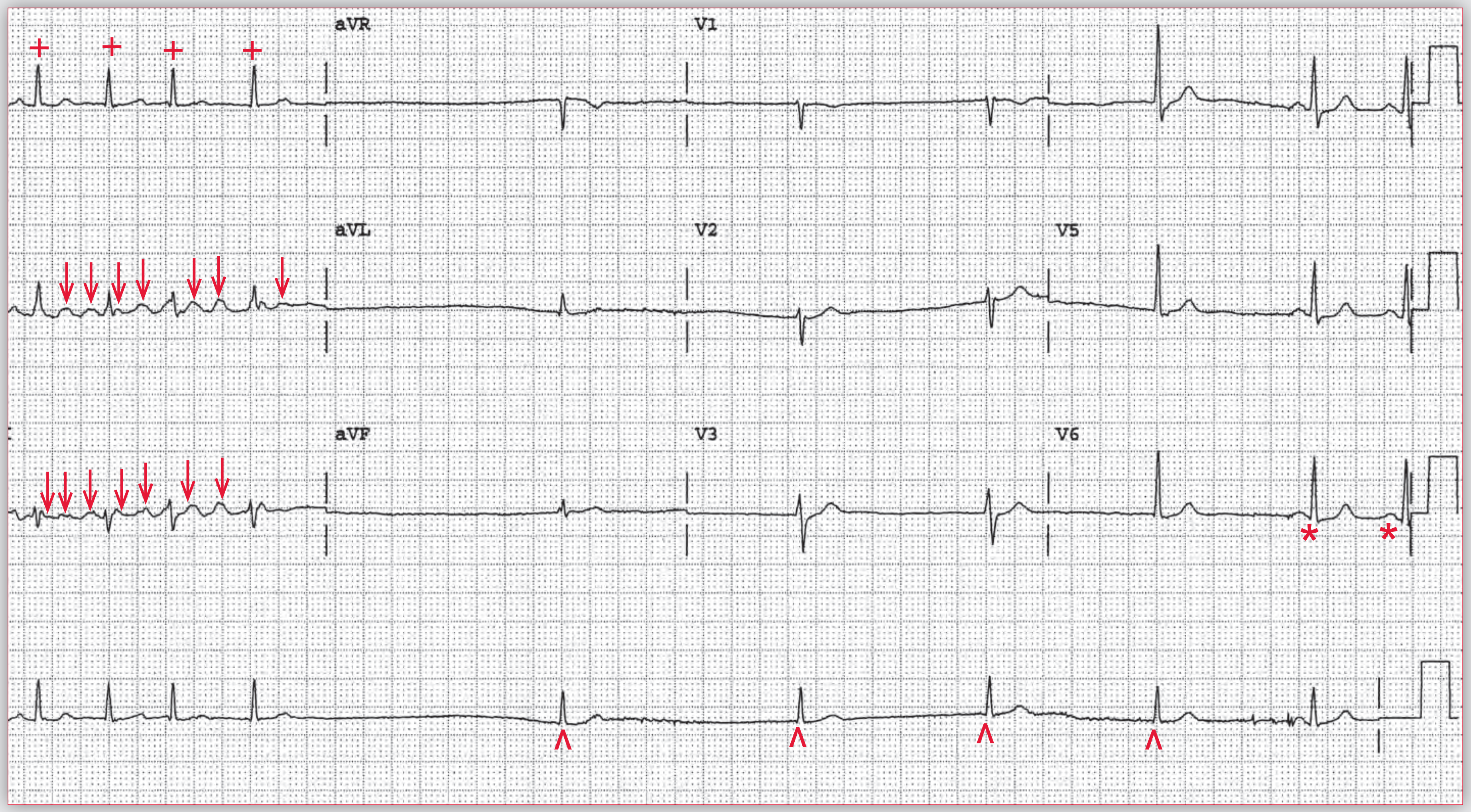
An 88-year-old man presents to his primary doctor with complaints of intermittent lightheadedness. He states that over the past 2 weeks, he has noted occasional episodes of profound lightheadedness that last a few seconds and resolves spontaneously. He denies any other symptoms, and a general review of symptoms is unremarkable.

As the primary doctor is recording an ECG, the patient becomes diaphoretic and states he is lightheaded.

What findings on the ECG explain the patient's symptoms?

What is the diagnosis for this condition?





ECG 106 Analysis: Atrial fibrillation, spontaneous termination with sinus arrest, junctional escape rhythm followed by sinus rhythm (sick sinus syndrome, tachycardia-bradycardia)

The first 4 QRS complexes (+) have a normal duration (0.08 sec) and the rhythm is irregularly irregular. The average rate is about 100 bpm. There are coarse and irregular waveforms (↓) seen between each QRS complex. Therefore, this is atrial fibrillation. The QRS complex morphology is normal, although there is low voltage in the limb leads (QRS complex < 5 mm in each lead). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (300/390 msec).

After the fourth QRS complex, there is an abrupt termination of the atrial fibrillation, and after a pause of 2.2 sec there is an escape rhythm with narrow QRS complexes (^) that have the same morphology as the initial QRS complexes during atrial fibrillation. There is no atrial activity seen before or after these QRS complexes. Hence complexes 5–8 are junctional complexes and this is an escape junctional rhythm. The rate initially is 38 bpm and slowly increases to a rate of 50 bpm. The gradual increase in the junctional rate is seen with an ectopic junctional rhythm. The last two complexes have P waves (*) before them with a stable PR interval (0.16 sec). These are two sinus beats at a rate 96 bpm. Hence there is a 7.4-sec pause after the abrupt termination of the atrial fibrillation before sinus rhythm develops. This is a manifestation of a sick sinus syndrome and is termed a tachycardia-bradycardia variant.

A sick sinus syndrome involves the pacemaker tissue and may also involve the AV node. In this case, the AV node was intact as there was an escape junctional rhythm. However, if there were AV nodal involvement without a junctional escape rhythm, there would have been a 7.4-sec period of asystole before resumption of a sinus impulse, which could have resulted in syncope. Indeed, it is the long offset pause after an atrial tachyarrhythmia that is the etiology for the syncope seen with a sick sinus syndrome.

The sick sinus syndrome, which may also involve the AV node, has various manifestations:

1. Tachycardia-bradycardia syndrome, in which an atrial tachyarrhythmia, most often atrial fibrillation, terminates abruptly and is followed by a variable period of a bradycardia or even asystole. During this time there is absence of sinus node activity. This diagnosis is established when there is a temporal relationship between the tachycardia and bradycardia, *ie*, bradycardia immediately follows the tachycardia.
2. Bradycardia-tachycardia syndrome, in which a profound bradycardia (sinus or junctional) is followed by the occurrence of an atrial tachyarrhythmia that is an escape rhythm. There is a temporal relationship between the bradycardia and tachycardia, *ie*, the tachyarrhythmia immediately follows the bradycardia.
3. A profound sinus bradycardia with chronotropic incompetence, *ie*, a failure of the sinus rate to respond appropriately to an increase in catecholamines or sympathetic stimulation as occurs with exercise.
4. A premature atrial complex followed by a very long pause until there is resumption of a sinus complex.

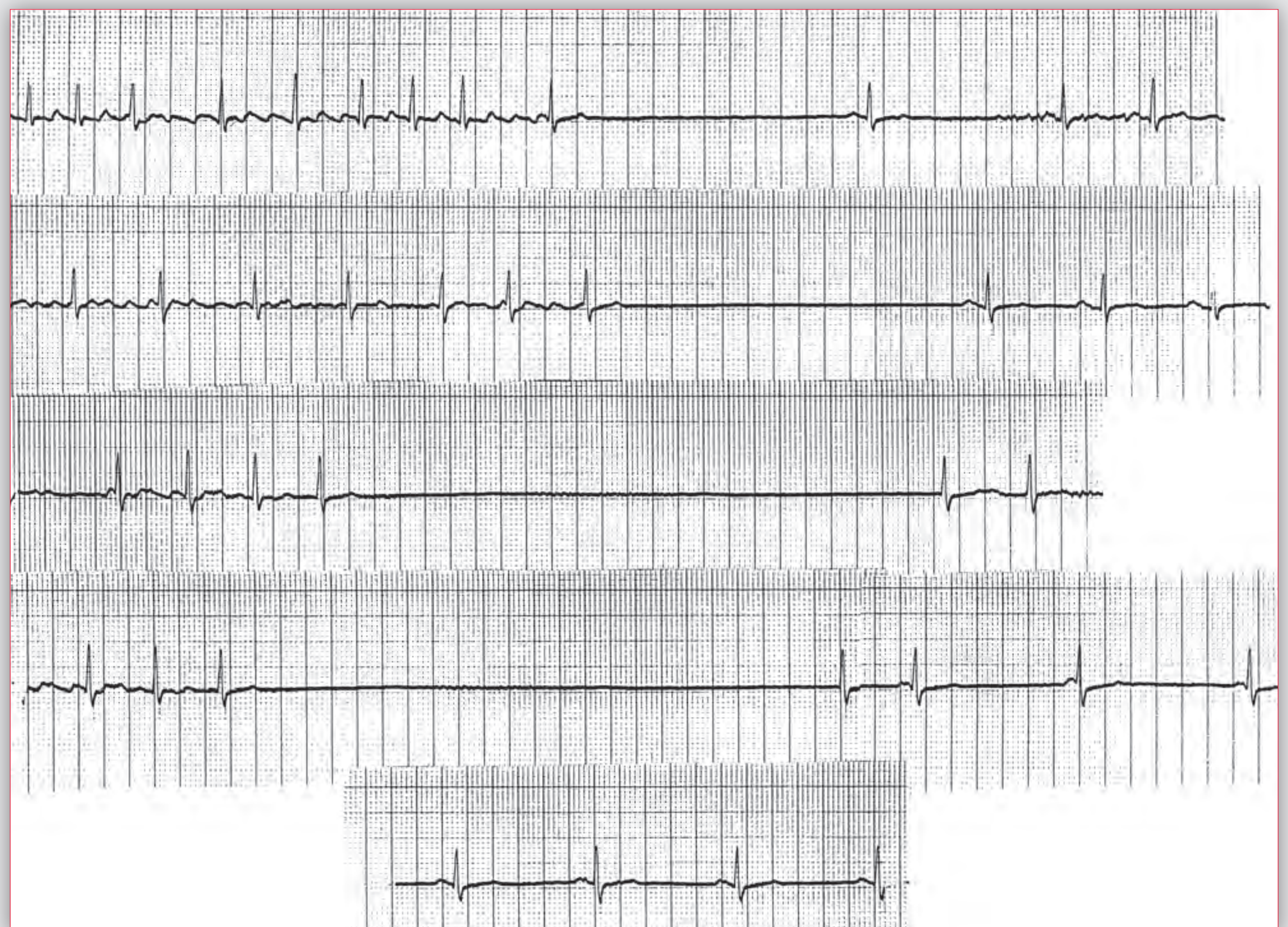
continues

5. The development of permanent atrial fibrillation (the result of sinus node dysfunction) with a slow ventricular response rate in the absence of AV nodal blocking agents. This reflects concomitant AV nodal involvement.
6. Sinus node arrest, *ie*, a long RR interval or pause with the absence of a P wave seen during this interval. The PP interval surrounding the pause is longer than two sinus PP intervals.

When the sick sinus syndrome is associated with symptoms of a bradycardia, *ie*, syncope or presyncope with an offset pause or fatigue and lightheadedness associated with a persistent bradycardia, the treatment is a pacemaker. For patient with evidence of sinus node function, a dual-chamber pacemaker is inserted; the pacemaker is programmed for mode switching so that the occurrence of atrial fibrillation does not cause tracking by the pacemaker and the occurrence of rapid paced rates. Patient who are in permanent atrial fibrillation with a slow ventricular response rate generally receive a single-lead pacemaker programmed for VVI pacing.

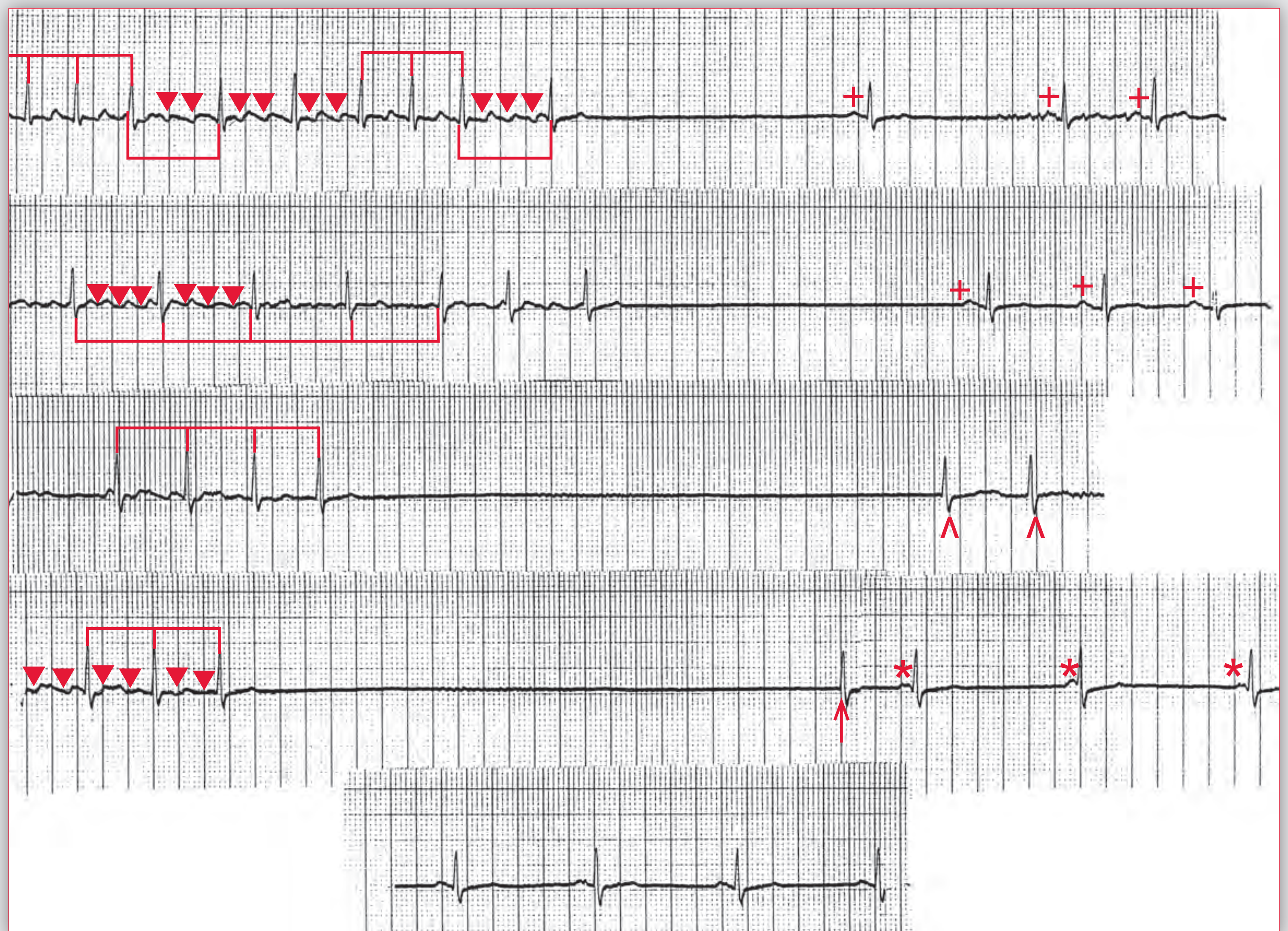
An alternative approach to the therapy of a tachycardia-bradycardia syndrome is to prevent the atrial tachyarrhythmia with the use of an antiarrhythmic agent, *ie*, class IA, IC, or III. Preventing the atrial arrhythmia will prevent the offset pause and bradycardia that follows. ■

A 76-year-old woman presents to the emergency department with complaints of palpitations that are associated with episodes of lightheadedness and a sensation that she is about to lose consciousness. Her physical examination is unremarkable and her ECG shows normal sinus rhythm. She is admitted to the hospital and placed on telemetry. Later in the day, she complains of lightheadedness and has a transient period of loss of consciousness. The telemetry monitoring strips recorded during this event are presented.



What arrhythmia is present?

What is the cause of her symptoms?



ECG 107 Analysis: Atrial flutter with abrupt termination, offset pause with resumption of normal sinus rhythm, sick sinus syndrome (tachycardia-bradycardia)

Presented are a series of telemetry rhythm strips. The first and second rhythm strips shows an initial rhythm that is regularly irregular, *ie*, although irregular, the long RR intervals (⊔) short RR intervals (⊓) are the same. Although there is no organized atrial activity seen, there are regular undulations of the baseline (▼), which are uniform in morphology and are occurring at a regular rate of 300 bpm. Hence the underlying rhythm is atrial flutter with variable AV block. The QRS complexes are narrow and hence they are supraventricular. After the tenth complex, there is an abrupt termination of the atrial flutter and after a pause of 2.4 sec (on strip 1) and 3 sec (strip 2), there are narrow QRS complexes that are preceded by a P wave (+) with a constant PR interval (0.20 sec). This is a sinus rhythm with a rate that gradually increases to 86 bpm. There is no evidence of atrial activity during the pause. The third rhythm strips also initially shows atrial flutter, which abruptly terminates. After a pause of 4.8 sec, there are QRS complexes without evidence of atrial activity (^). Hence there is an escape junctional rhythm. During the pause, there is no evidence of atrial activity. The fourth strip also shows atrial flutter as the initial rhythm. This arrhythmia abruptly terminates and there is a pause of 4.8 sec. There is no atrial activity seen during the pause. Ending the pause is a narrow QRS complex (↑) without a preceding P wave. This is a junctional complex. The following three narrow QRS complexes have P wave preceding them (*) but the PR intervals are variable. Hence there is resumption of a sinus rhythm, but the QRS complexes are no the result of the atrial activity, *ie*, these are junctional complexes. The last rhythm strip shows normal sinus rhythm with a stable PR interval (0.20 sec), which is identical to the PR intervals of the sinus complexes seen on the other rhythm strips.

The diagnosis in this patient is a sick sinus syndrome, tachycardia-bradycardia variant. The atrial arrhythmia is atrial flutter that abruptly terminates to a sinus or junctional rhythm. As the offset pauses are associated with symptoms, this patient requires a permanent pacemaker.

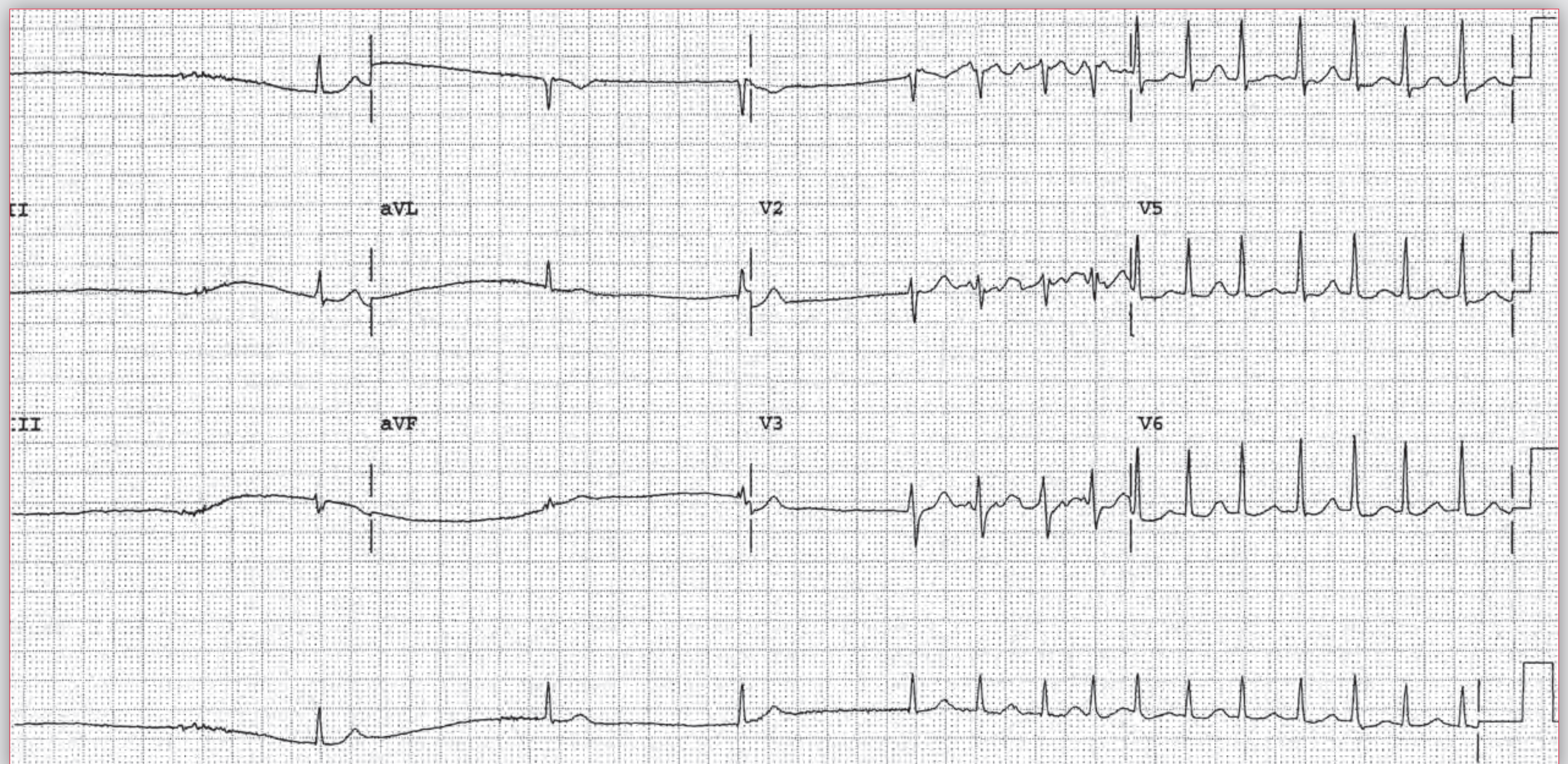
An alternative approach to the therapy of a tachycardia-bradycardia syndrome is to prevent the atrial tachyarrhythmia with the use of an antiarrhythmic agent, *ie*, class IA, IC, or III. Preventing the atrial arrhythmia will prevent the offset pause and bradycardia that follows. ■

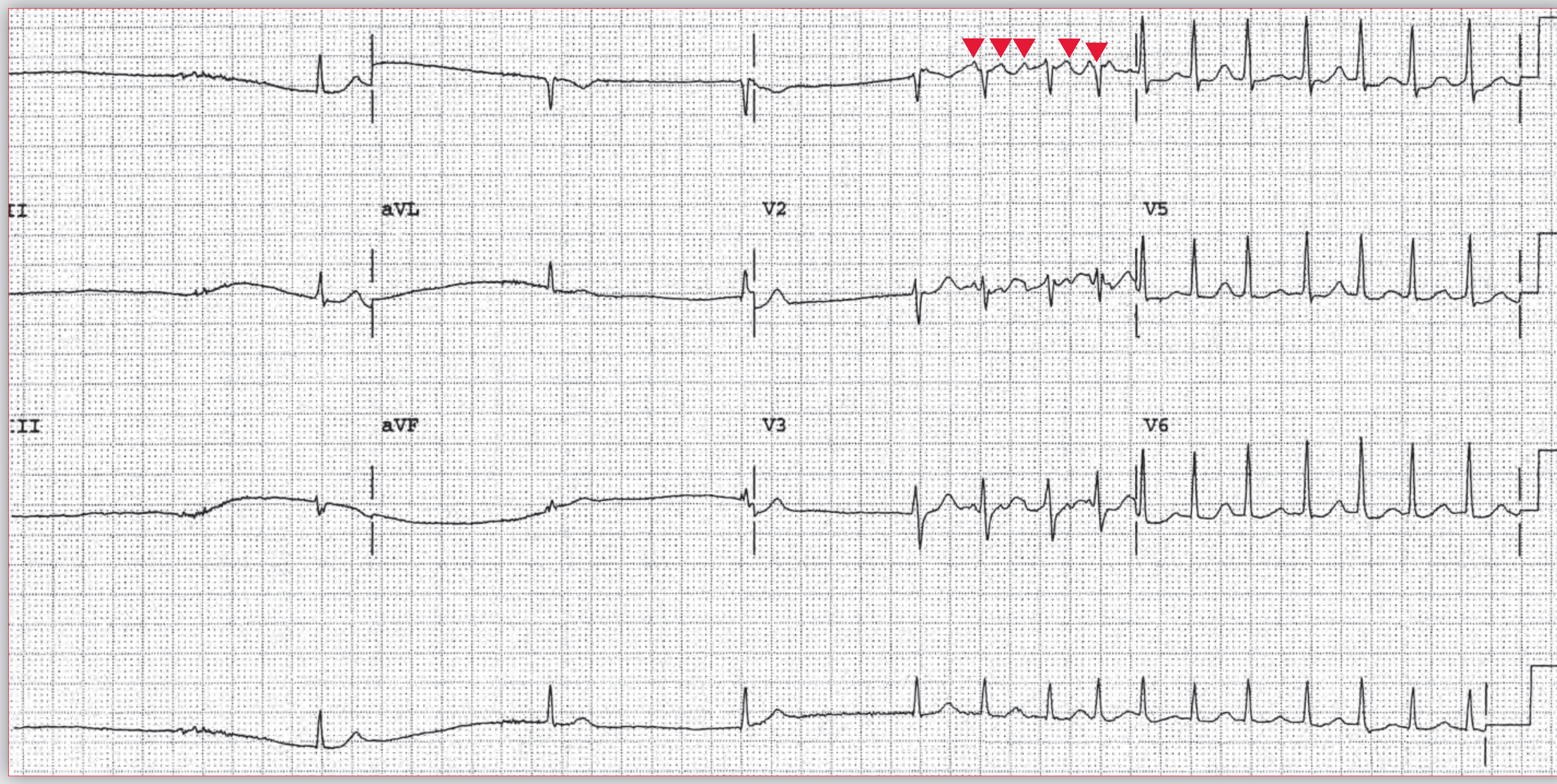
Notes

A 92-year-old female is brought to the local emergency department by her grandson, who found her on the floor of her kitchen when he went to visit her that evening. He states that she was "acting crazy" when he found her. Upon further questioning, he describes finding her supine on the floor without signs of trauma or a fall (no overturned furniture or fallen objects, no obvious bodily injury). The patient was semi-conscious. He activated emergency medical services when she was not able to properly answer his questions.

She is brought into the acute care bay in the emergency department and initial survey is remarkable for slow peripheral pulses that are difficult to palpate. She is attached to a cardiac monitor. Shortly thereafter, much to the surprise of the medical staff, she suddenly asks, "Where am I?" Her mental status appears to have spontaneously improved. The attending physician prints out an ECG from the monitor corresponding to the moment of her clinical improvement.

What explanation for the patient's condition does the ECG provide?





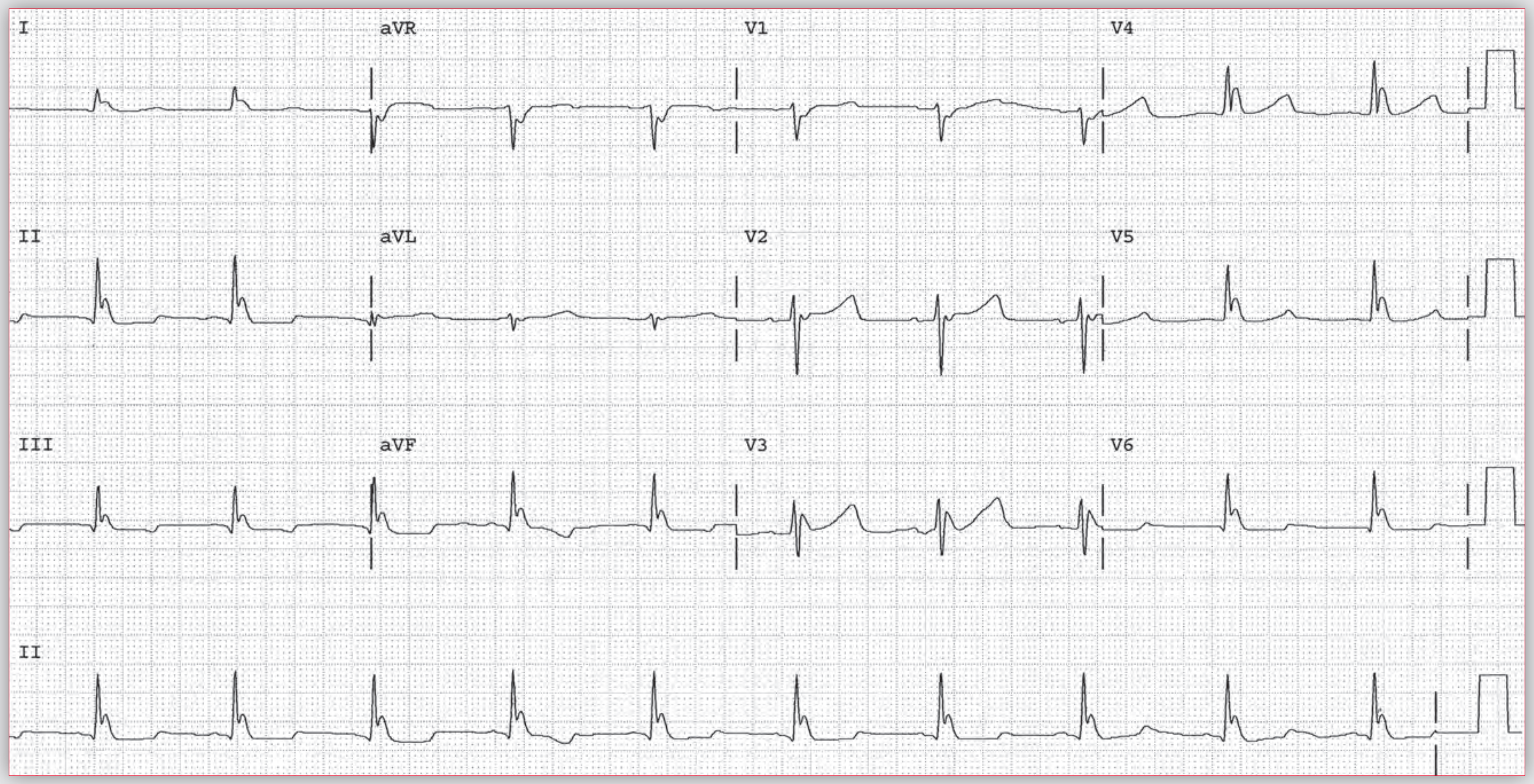
ECG 108 Analysis: Junctional rhythm followed by atrial fibrillation with rapid ventricular response, sick sinus syndrome (bradycardia-tachycardia)

The initial portion of the ECG shows QRS complexes with normal duration (0.08 sec). They have a normal morphology, although there is low voltage in the limb leads (QRS < 5 mm in each lead). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). There are no P waves seen before or after any of these QRS complexes. Therefore, this is a junctional rhythm. The rate is variable, reflecting the fact that this is from an ectopic junctional focus that is unstable and has a variable rate of depolarization. After the first 3 QRS complexes, the rate become faster and is irregularly irregular. Also noted are rapid and irregular undulations (▼) of the baseline, reflect atrial activity. This is atrial fibrillation and the ECG represents a manifestation of a sick sinus syndrome known as a bradycardia-tachycardia variant. As a result of the slow rate, there is an escape atrial tachyarrhythmia. Hence there is a temporal relationship between the two rhythms, *ie*, a tachycardia immediately follows the bradycardia. ■

Core Case 109

A 48-year-old man is found unconscious on a park bench. He is brought to the emergency department, where an ECG is obtained. A repeat ECG is obtained about 4 hours after admission and therapy (ECG 109B).

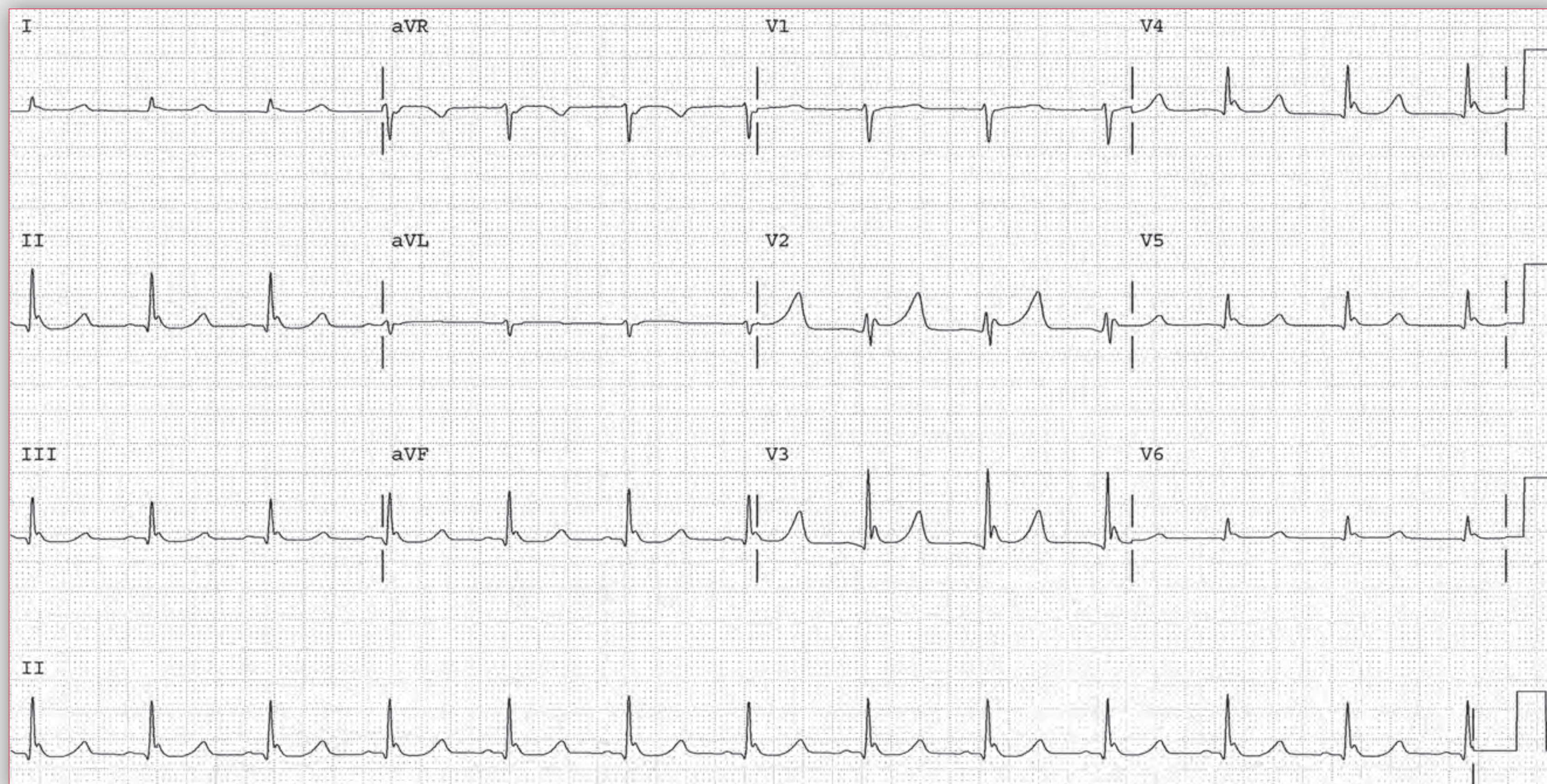
ECG 109A

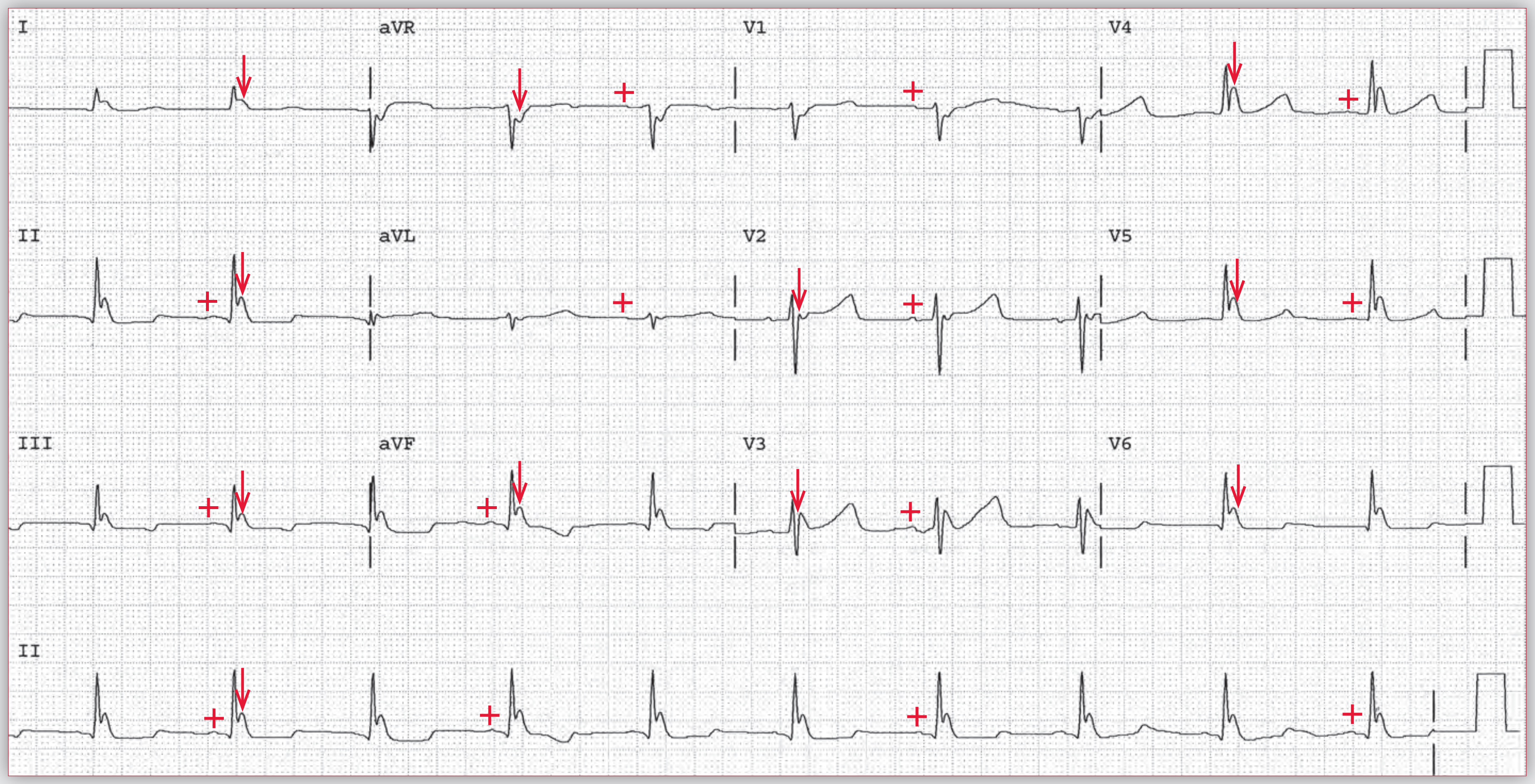


Is the ECG 109A normal?

If not, what is the abnormality noted and what likely mechanism for the abnormality?

ECG 109B





ECG 109A Analysis: Sinus rhythm with marked J-point elevation (Osborn or J waves)

ECG 109A shows a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm.

The QRS complex duration is normal (0.10 sec). The axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are slightly prolonged (480/480 msec). Following the QRS complex, at the J point, is an abnormal waveform (↓) that is significantly elevated. This is termed a J wave or Osborn wave, and it is the result of hypothermia that may be due to exposure, inadequate warming after cardiac surgery, or therapeutic hypothermia in sudden cardiac death survivors. Upon admission to the emergency department, this patient's temperature was 89°F. In addition to the Osborn wave, hypothermia is also associated with prolongation of the QT interval.

The normal ventricular myocardium has three electrophysiologically distinct layers of cells: epicardial, endocardial, and M cells. These three cell types have different electrophysiologic properties, primarily related to the rate of repolarization and hence action potential duration. The rate of repolarization can be altered by temperature changes; this especially affects the epicardial and M cells. Therefore, with hypothermia there is an increase in the dispersion of repolarization across the ventricular wall, accounting for the Osborn wave. This abnormality of early repolarization is also seen in the Brugada pattern, which is congenital resulting from a genetic abnormality of the SNC5A gene. The J point abnormality seen with Brugada pattern is over the right ventricular precordial leads (*ie*, V1–V3). Temperature changes, such as hyperthermia, can also affect the Brugada pattern, making it more pronounced.

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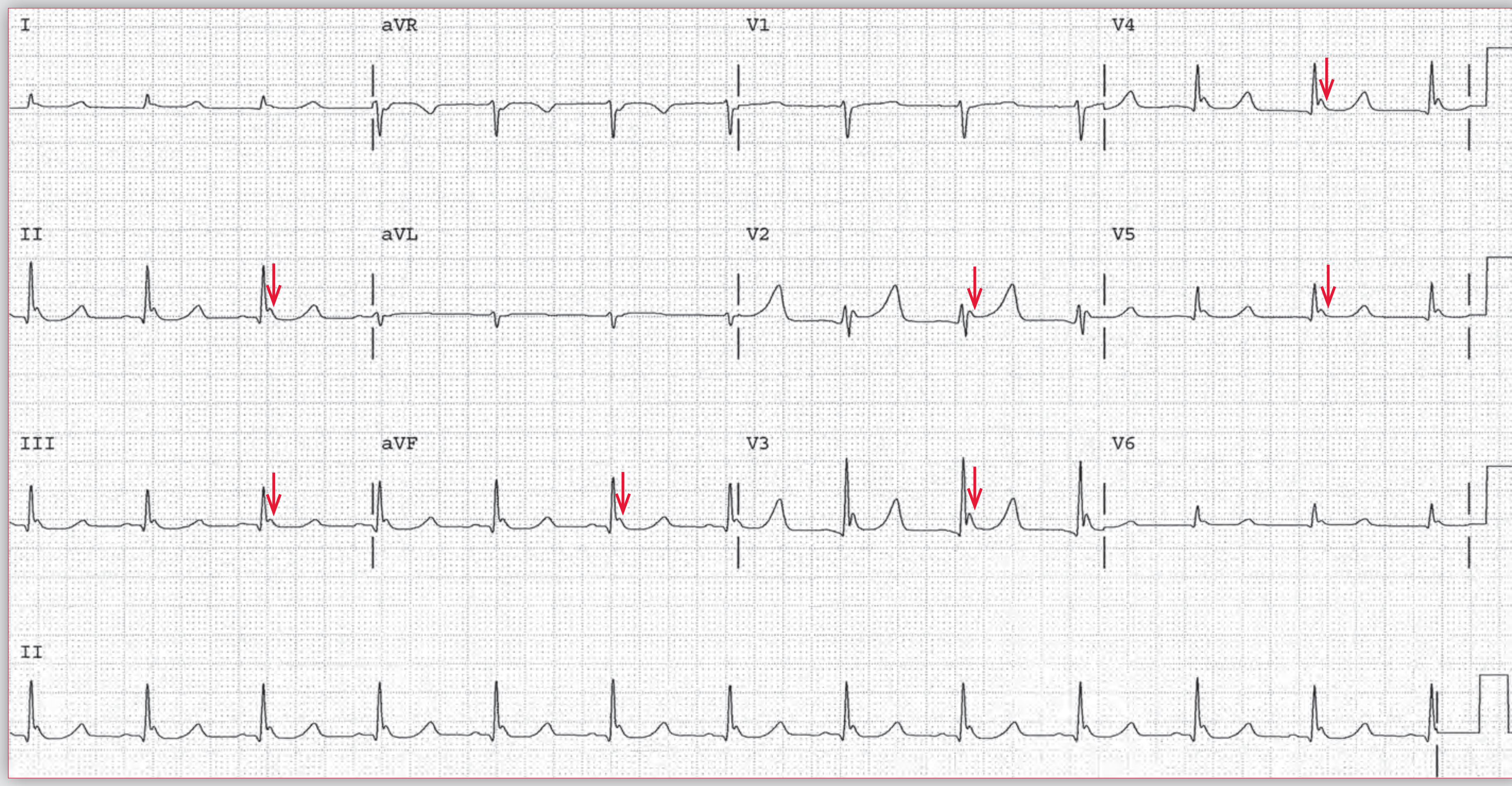


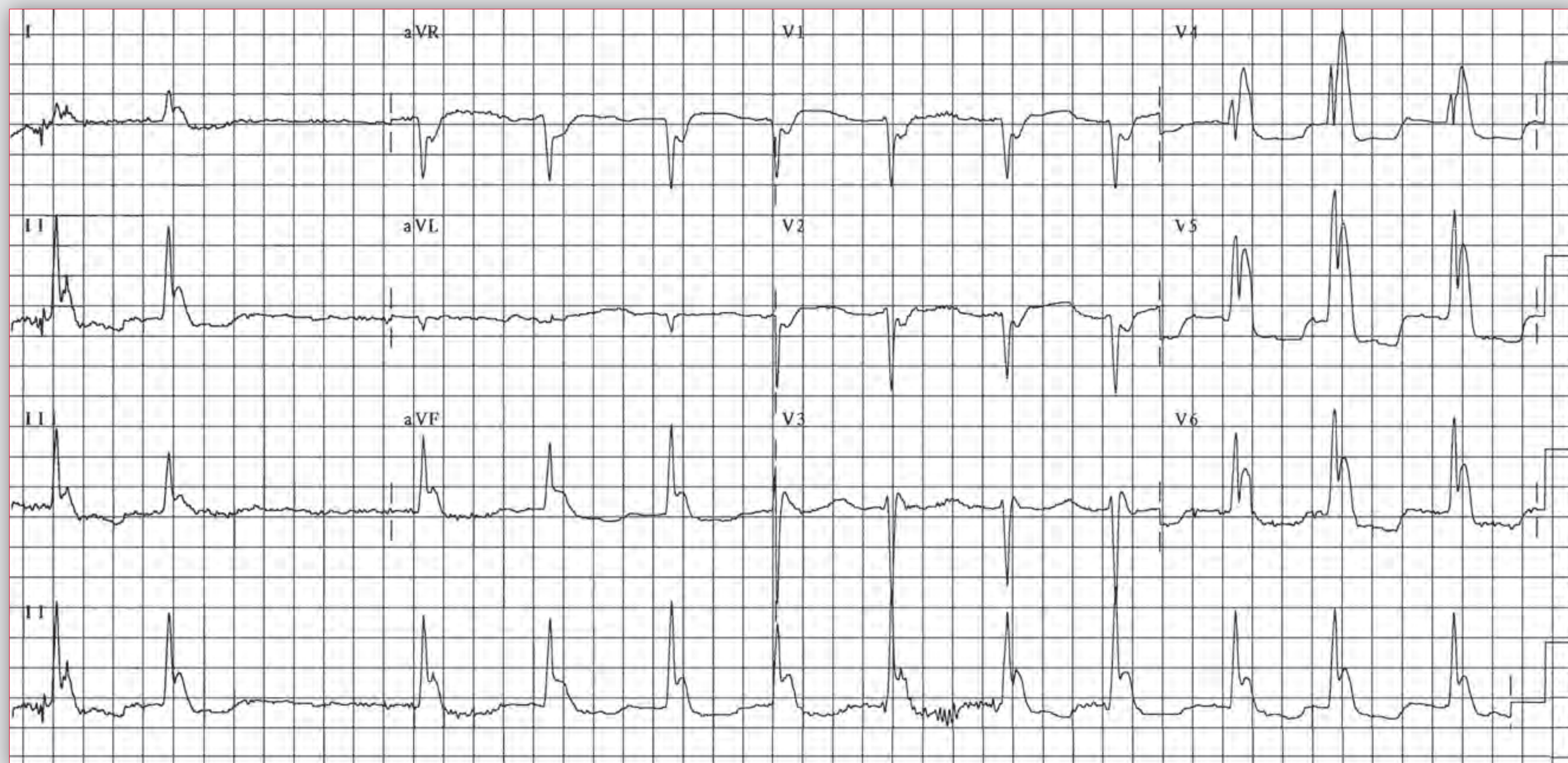
ECG 109B Analysis: Sinus rhythm with less marked Osborn waves

ECG 109B is from the same patient as ECG 109A. The patient's temperature was 93°F. There is a sinus rhythm at a rate of 76 bpm. The QRS complex duration, axis, and PR interval are the same as seen in ECG 109A. The QT/QTc are shorter and near normal (400/450 msec). Although Osborn waves (↓) are still present, their height is less as a result of a warmer temperature. There is a relationship between the temperature and the height of the Osborn (*ie*, the lower the temperature, the taller and more prominent are Osborn waves). ■

Core Case 110

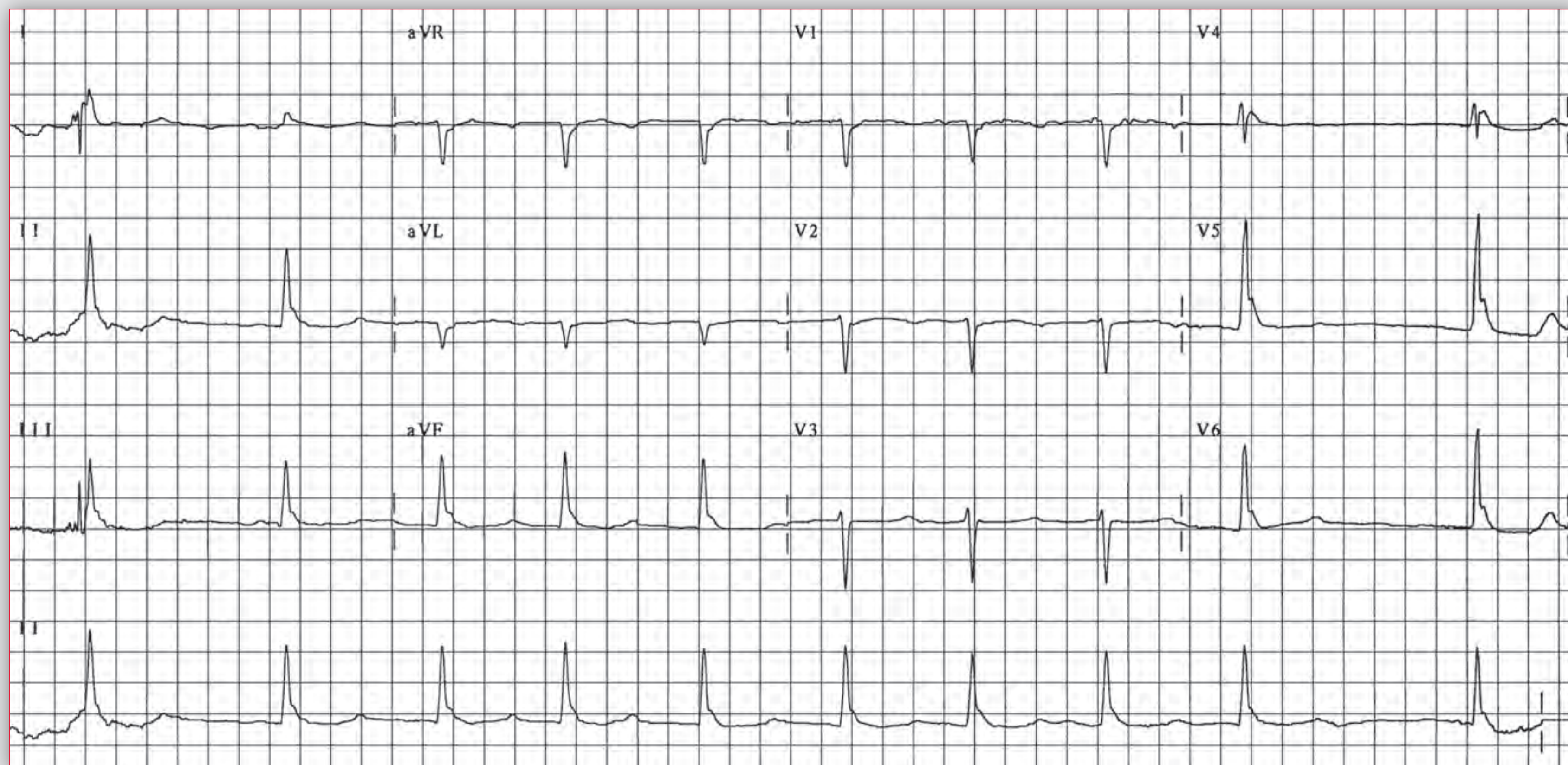
A 28-year-old man who was reported missing during a snowshoe trek through the Rocky Mountains was found 3 days later. He was brought by helicopter to the closest hospital. Upon arrival his body temperature was 79.2°F.

ECG 110A



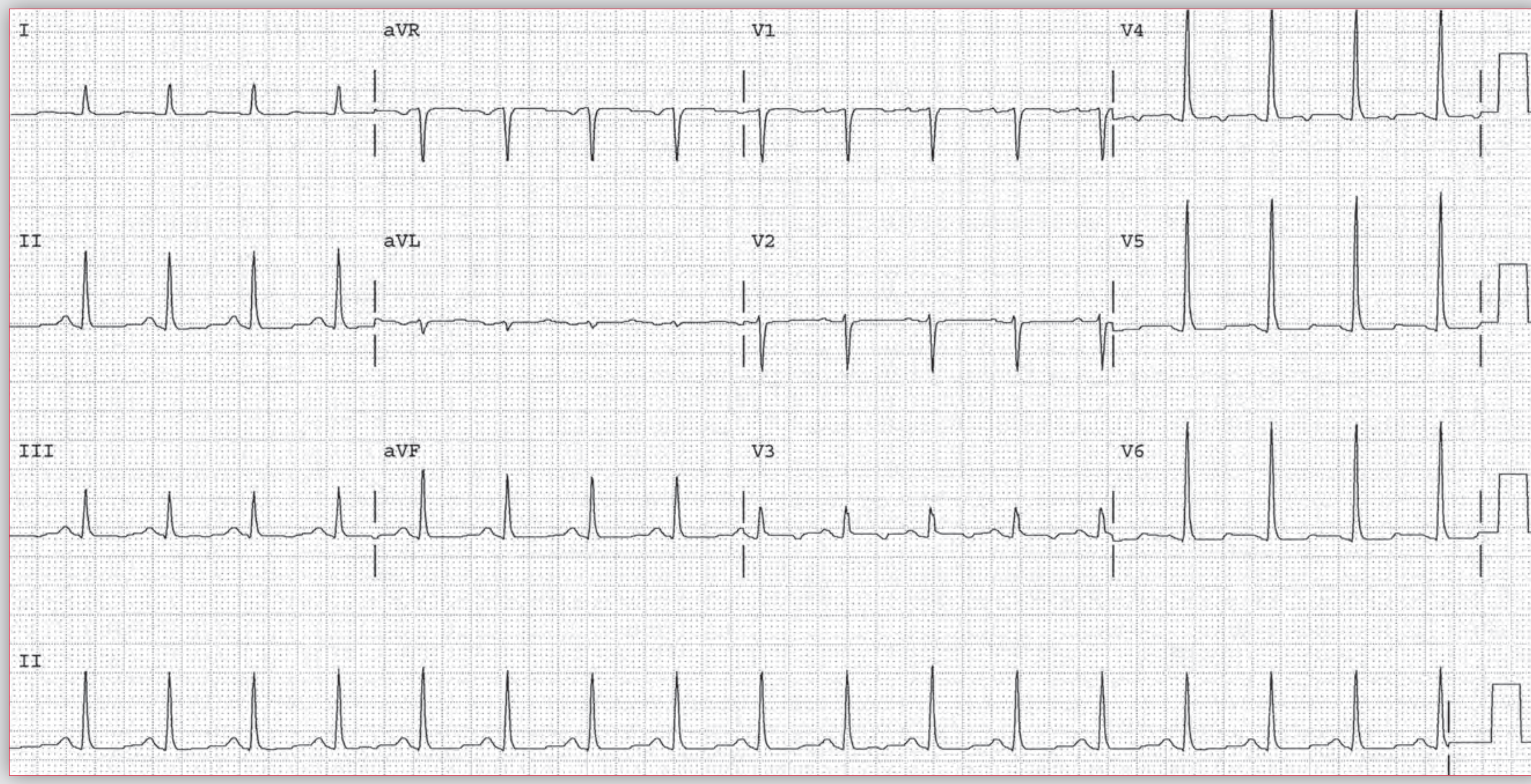
An ECG was obtained (ECG 110A). One hour later, his body temperature was 82.6°F and an ECG was repeated (ECG 110B). On the following day, his body temperature was normal and ECG 110C was obtained.

ECG 110B

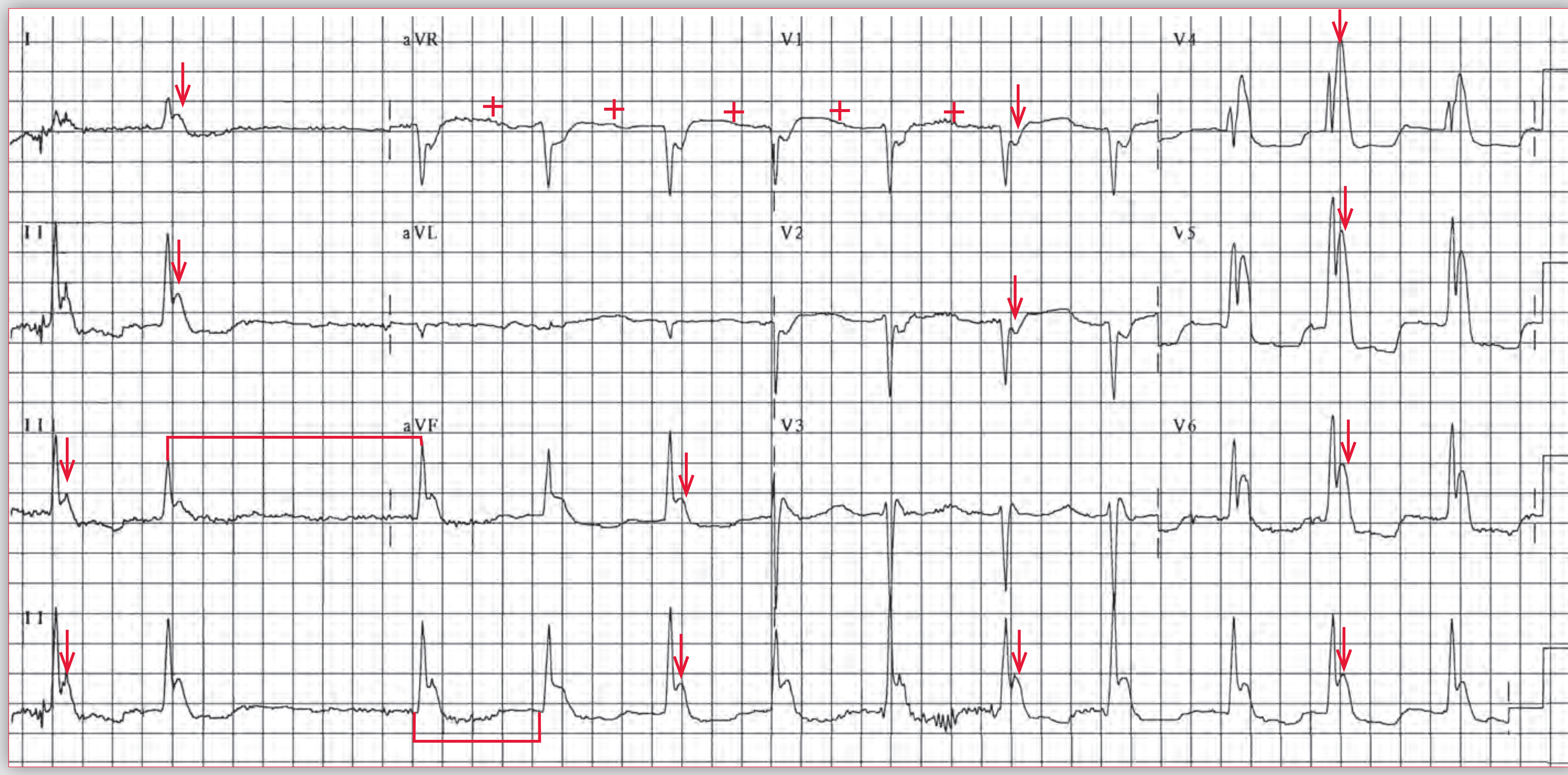


Core Case 110

ECG 110C



What is the principal abnormality seen on these ECGs?

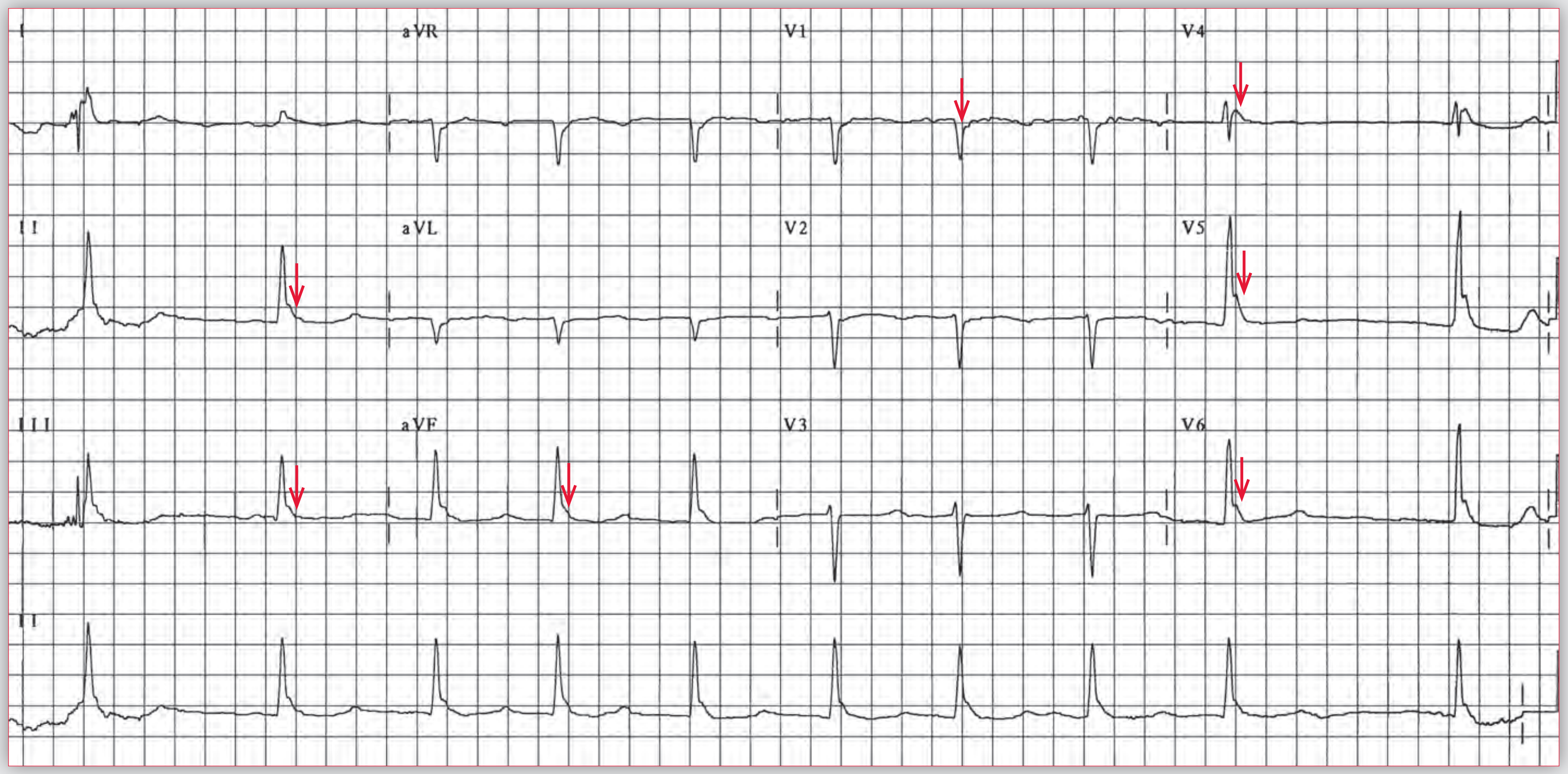


ECG 110A Analysis: Sinus rhythm, sinus node exit block, marked J point elevation (Osborn waves), and QT prolongation

ECG 110A shows there is a regular rhythm at an average rate of 74 bpm. There is one long RR interval noted (⊓). In addition, the eleventh QRS complex (^) is slightly premature. P waves (+) can be seen before each QRS complex, although they are very small and barely obvious. The PR interval is constant (0.32 sec). Therefore, this is probably a normal sinus rhythm with a first-degree AV block. The eleventh complex is likely a premature atrial complex. The long RR interval (⊓) is equal to two RR intervals (⊔); this is a sinus node exit block.

The QRS complex duration is normal (0.08 sec). The axis is normal between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are prolonged (520/580 msec). Noted are prominent waveforms (↓) after the QRS complexes that are significantly elevated and wide. These are J waves or Osborn waves. The Osborn waves and long QT are characteristic of hypothermia.

continues



ECG 110B Analysis: Atrial fibrillation, Osborn waves (slightly less prominent), QT prolongation

ECG 110B is from the same patient as ECG 110A. The rhythm is irregularly irregular with a rate of 62 bpm. There are no obvious P waves seen. Hence the rhythm is atrial fibrillation. The QRS complex duration and axis are the same as in ECG 110A. Osborn waves (↓) can still be seen, but they are less prominent as a result of a higher body temperature. The QT/QTc intervals are still long (500/530 msec), but they are slightly shorter than noted on the first ECG.

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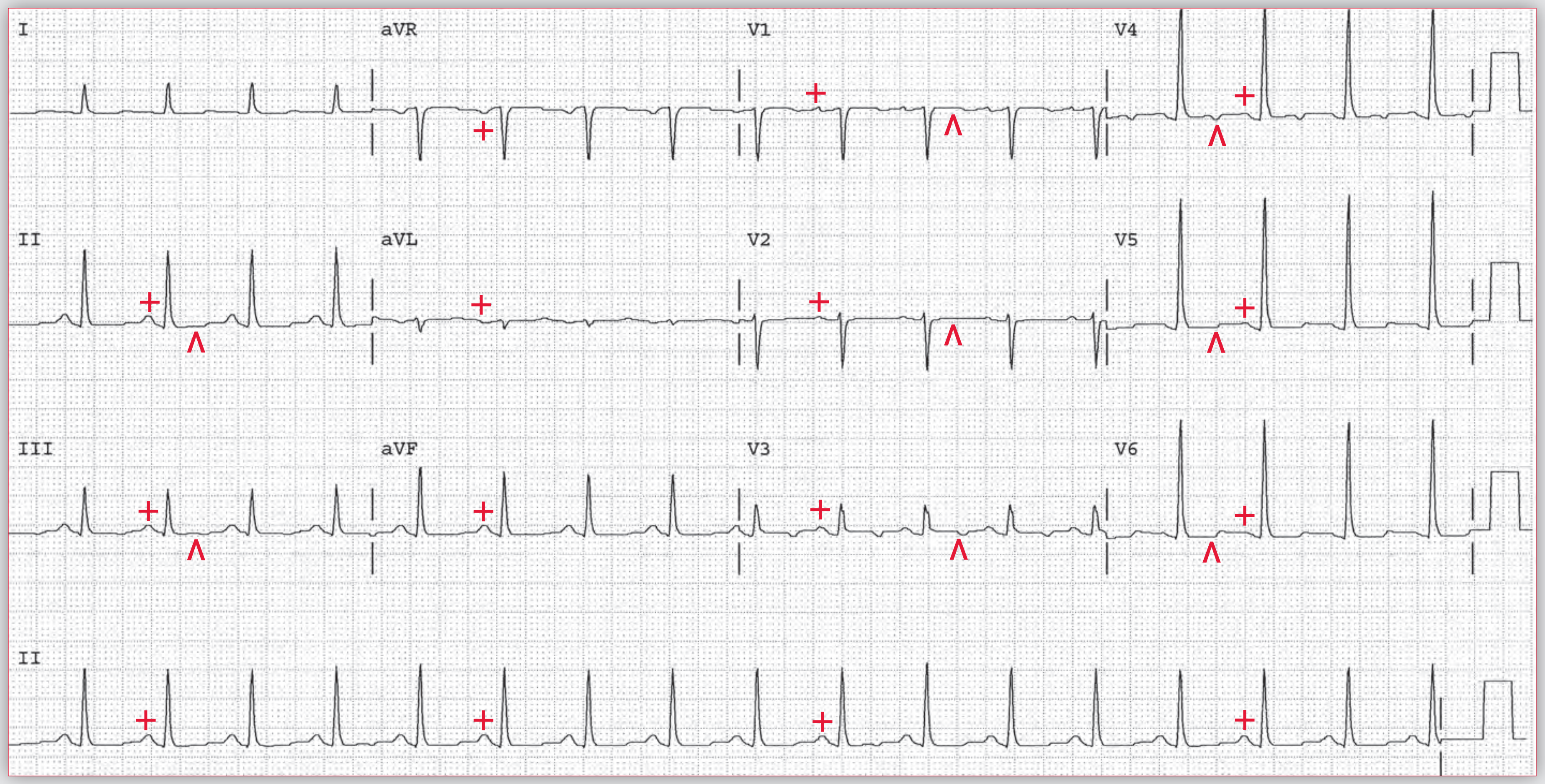


ECG 110C Analysis: Sinus tachycardia, nonspecific ST-T wave changes.

ECG 110C is from the same patient as ECGs 110A and 110B. It was obtained when his body temperature was normal. There is a regular rhythm at a rate of 102 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (QRS positive in leads I and aVF). There are diffuse ST-T wave abnormalities (^) and the QT/QTc intervals are normal (340/440 msec). Osborn waves are no longer seen as the patient's temperature was normal.

The normal ventricular myocardium has three electrophysiologically distinct layers of cells: epicardial, endocardial, and M cells. These three cell types have different electrophysiologic properties, primarily related to the rate of repolarization and hence action potential duration. The rate of repolarization can be altered by temperature changes; this especially affects the epicardial and M cells. Therefore, with hypothermia there is an increase in the dispersion of repolarization across the ventricular wall, accounting for the Osborn wave. This abnormality of early repolarization is also seen in the Brugada pattern, which is a congenital condition due to an abnormality of the SNC5A gene. The J-point abnormality seen with Brugada pattern is over the right ventricular precordial leads (*ie*, V1–V3). Temperature changes (*ie*, hyperthermia) can also affect the Brugada pattern, making it more pronounced. ■

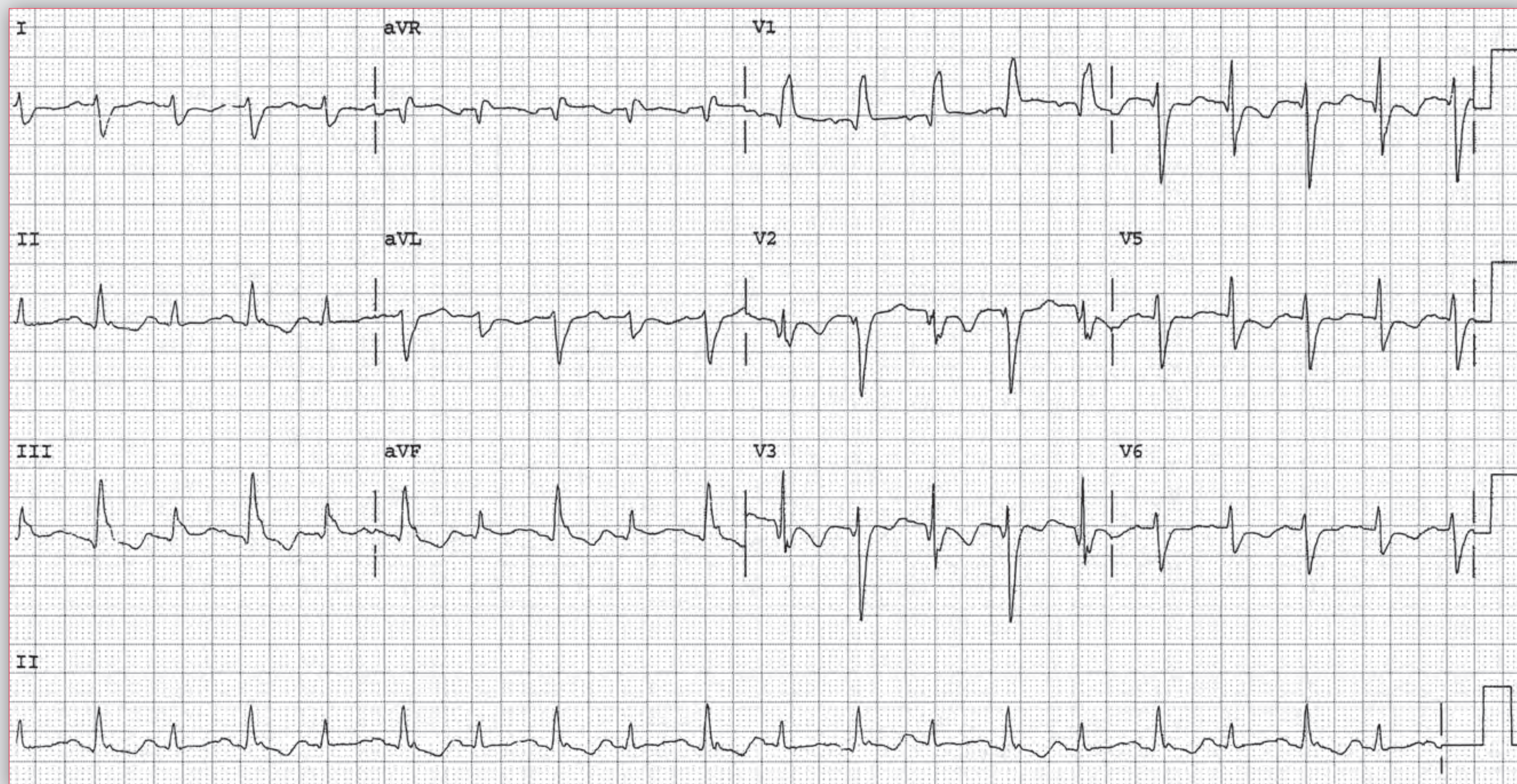
Notes

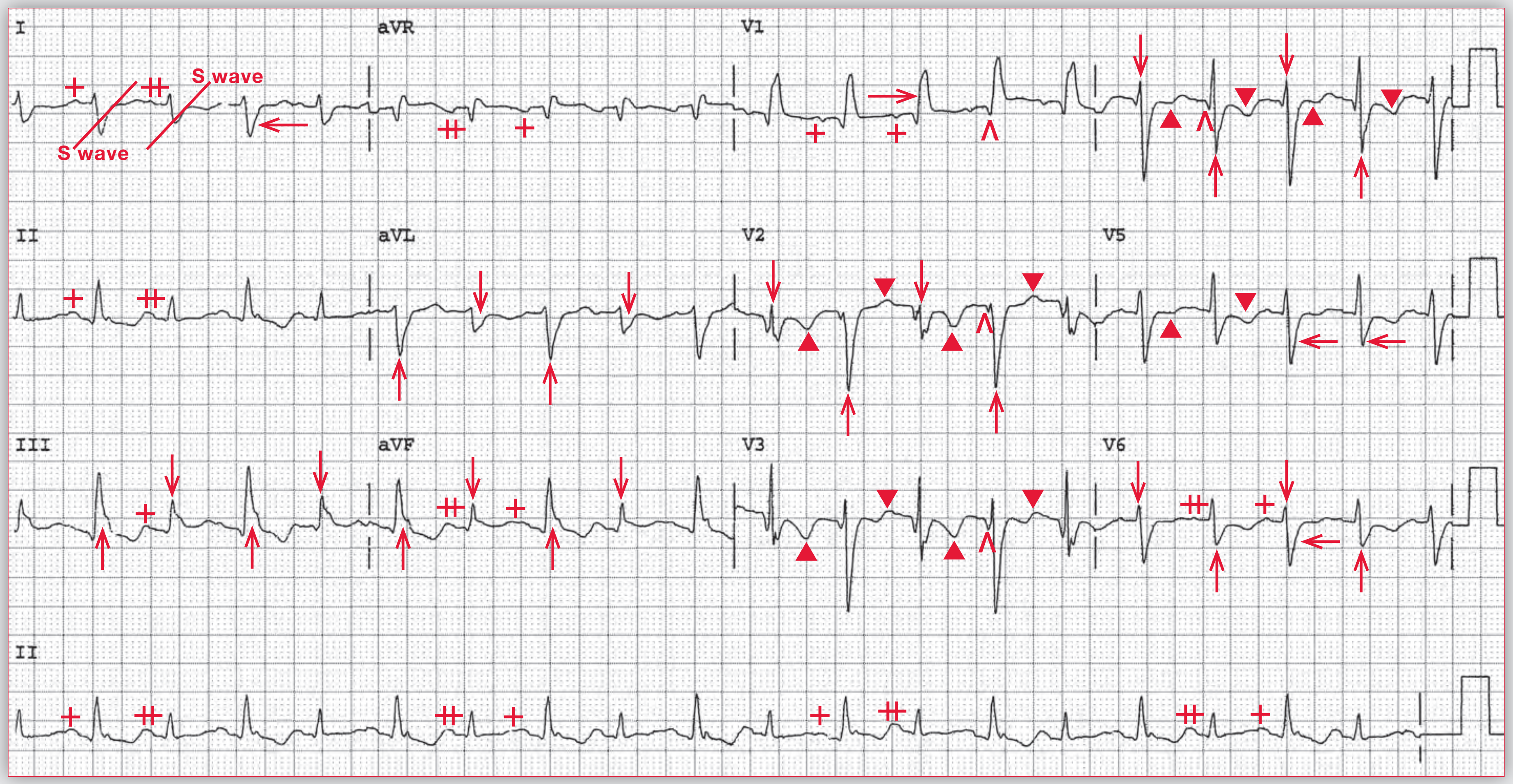
Core Case 111

A 48-year-old woman with systemic lupus erythematosis is admitted with pleuritic chest pain and lightheadedness. Her blood pressure is noted to be 80/60 and there is prominent neck vein distention. Heart sounds are distant. An ECG is obtained.

What does it show?

What is the likely etiology for her clinical presentation?





ECG 111 Analysis: Sinus tachycardia, right bundle branch block (RBBB), left posterior fascicular block, old anterior wall myocardial infarction, electrical alternans (QRS, T-wave, and P-wave alternans)

There is a regular rhythm at a rate of 124 bpm. There is a P wave (**+**,**++**) before each QRS complex with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complex duration is prolonged (0.16 sec) and there is a RBBB morphology with a broad terminal R wave in lead V1 (→) and broad S wave in leads I and V4–V6) (←). The axis is rightward between +90° and +180° (negative QRS complex in lead I and positive in lead aVF), even when the terminal S wave due to the RBBB is considered. There is no evidence for a lateral wall myocardial infarction (there is an rS morphology in leads I and aVL), right ventricular hypertrophy (which cannot be diagnosed in the presence of a RBBB), Wolff-Parkinson-White pattern (short PR and delta wave), right-left arm lead switch (negative P wave and T wave in leads I and aVL and positive P, QRS and T wave in lead aVR), or dextrocardia (resembling right-left arm lead switch and also with reverse R-wave progression). Hence this is a left posterior fascicular block. There are initial Q waves (^) in leads V1–V4, diagnostic of an old anterior wall myocardial infarction. The QT/QTc intervals are prolonged (360/520 msec) but are normal when the prolonged QRS complex duration is considered (300/430 msec).

Noted are beat-to-beat changes in QRS amplitude (↓, ↑); this is electrical or QRS alternans. Also noted are beat-to-beat changes in T wave (▲, ▼) as well as P-wave amplitude (**+**, **++**); hence there is T- and P-wave alternans.

Based on the presenting symptoms and the physical examination, the occurrence of electrical alternans in this case is due to a large pericardial effusion and cardiac tamponade, which causes a swinging of the heart with each beat in a fluid-filled pericardial sac (pendulum effect). Hence there are anatomic changes in the location of the heart. In this situation, there is QRS and T-wave alternans present as well as P-wave alternans.

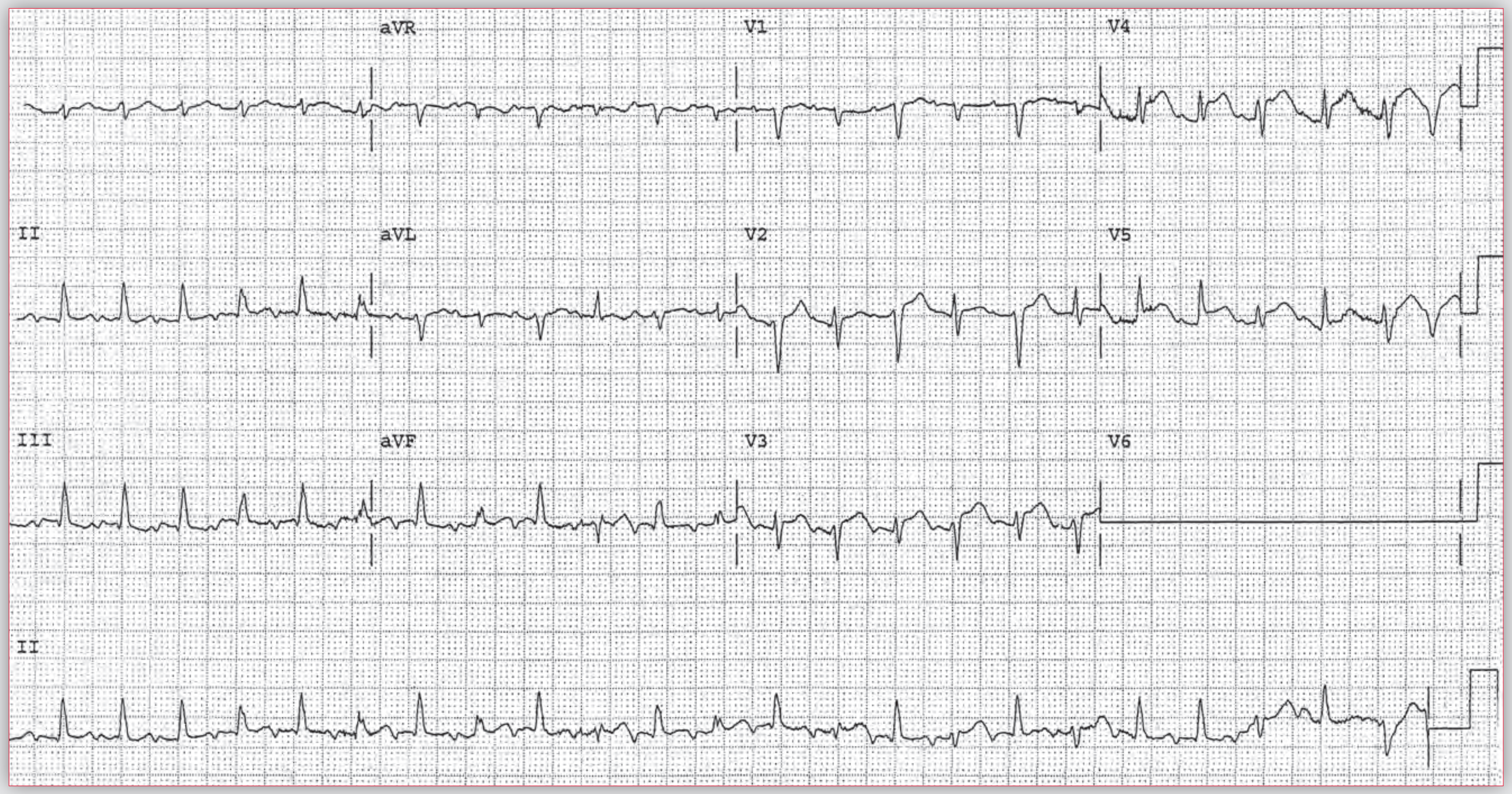
Electrical alternans, which can only be diagnosed when the rhythm is regular, may also be seen with other conditions, including any rapid, regular supraventricular tachycardia (atrial tachycardia, atrial flutter, atrioventricular nodal reentrant tachycardia [AVNRT], or atrioventricular reentrant tachycardia [AVRT]), associated with a preexcitation syndrome, an acute myocardial infarction, a dilated cardiomyopathy, or decompensated heart failure. In these situations, QRS and T-wave alternans are a result of beat-to-beat changes in calcium influxes into the ventricular myocardium. The atrial myocardium is not affected. As there is no change in the anatomic position of the heart, P-wave alternans is not seen in these situations. It has been reported that it may also be seen with ventricular tachycardia, but this is probably very uncommon. The forms of ventricular tachycardia in which it may be seen are a fascicular tachycardia or a right ventricular outflow tachycardia. ■

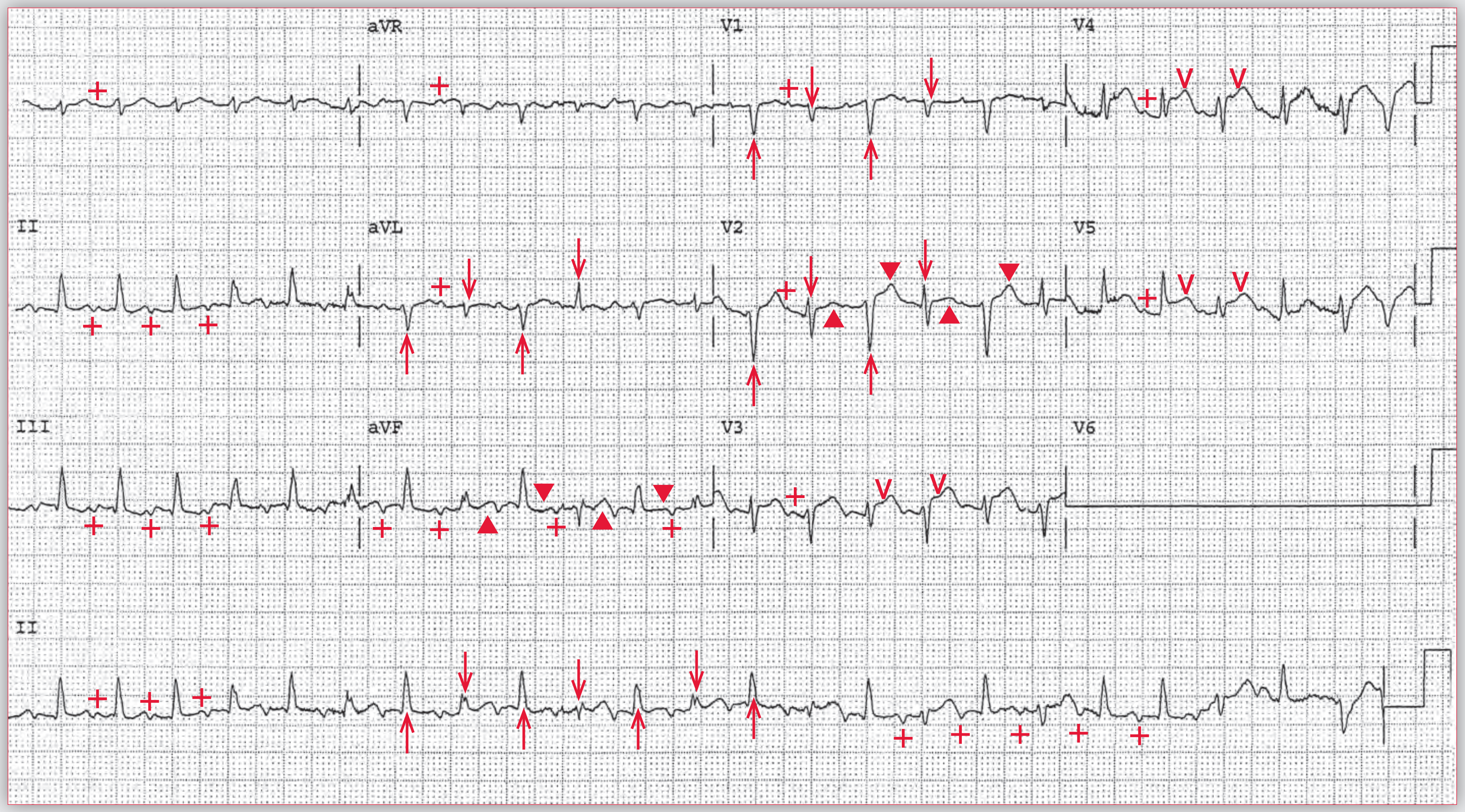
Notes

A 44-year-old man has a history of palpitations that have lasted for 3 hours. Associated with the palpitations is a chest burning that radiates to his throat. He comes to the emergency department, and an ECG is obtained.

What is the etiology of his palpitations?

What other abnormalities are noted on his ECG?





ECG 112 Analysis: Atrial tachycardia, electrical (QRS) alternans, T wave-alternans, low voltage, acute anterolateral myocardial infarction

There is a regular rhythm at a rate of 150 bpm. There are P waves (+) before each QRS complex, seen in most of the leads. The P waves are negative in leads II and aVF; hence this is not a sinus mechanism. There is a stable PR interval (0.16 sec) and RP interval (0.24 sec). This is a long-RP tachycardia. Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atypical or uncommon atrioventricular nodal reentrant tachycardia (AVNRT), due to conduction to the ventricle via the fast pathway and retrograde conduction to the atria via the slow pathway (fast-slow), or an atrioventricular reentrant tachycardia (AVRT). The P waves are negative in leads II and aVF, eliminating sinus tachycardia as an etiology. A second atrial waveform is not seen, making atrial flutter with 2:1 AV block unlikely. Unfortunately, the etiology for this arrhythmia cannot be definitively established on the ECG. Although an atypical AVNRT or AVRT are possible, a more common etiology for a long-RP tachycardia is an atrial tachycardia.

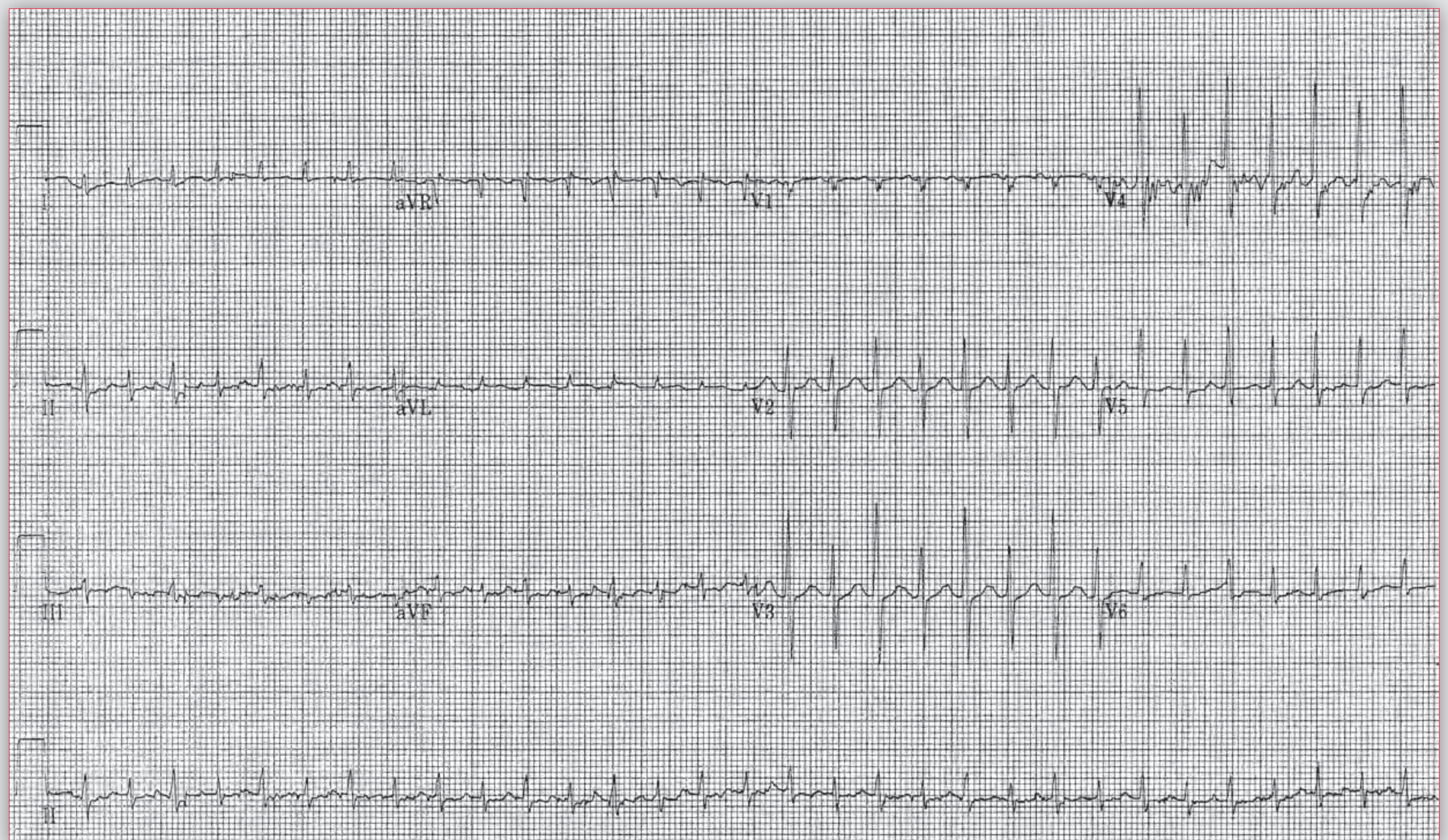
The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (QRS positive in leads I and aVF). The QRS morphology is normal, although there is low QRS voltage in all the leads (*ie*, QRS amplitude < 5 mm or little boxes in each limb lead and < 10 mm or little boxes in each precordial lead). The QT/QTc intervals are normal (280/440 msec). During the atrial tachycardia there is beat-to-beat variability in the amplitude of the QRS complex (↓, ↑) as well as beat-to-beat changes in the T wave (▲, ▼). This is QRS or electrical alternans and T-wave alternans; in this case, the alternans is due to a rapid, regular supraventricular tachycardia and is a result of beat-to-beat changes in calcium influxes. Also noted is ST-segment elevation in leads V3–V5 (∨), consistent with an acute anteroapical and anterolateral myocardial infarction. This may also be the etiology for electrical alternans. It is not clear if the atrial tachycardia has precipitated an acute myocardial infarction or if the two events are unrelated. Atrial arrhythmias are not precipitated by acute myocardial ischemia or myocardial infarction, unless there is evidence for left-sided heart failure or pericarditis. Nevertheless, the atrial arrhythmia should be treated acutely with rate slowing, and a β-blocker would be the drug of choice for its AV nodal blocking effect as well as for the acute myocardial infarction. An urgent catheterization and revascularization is indicated, regardless of the rhythm abnormality. ■

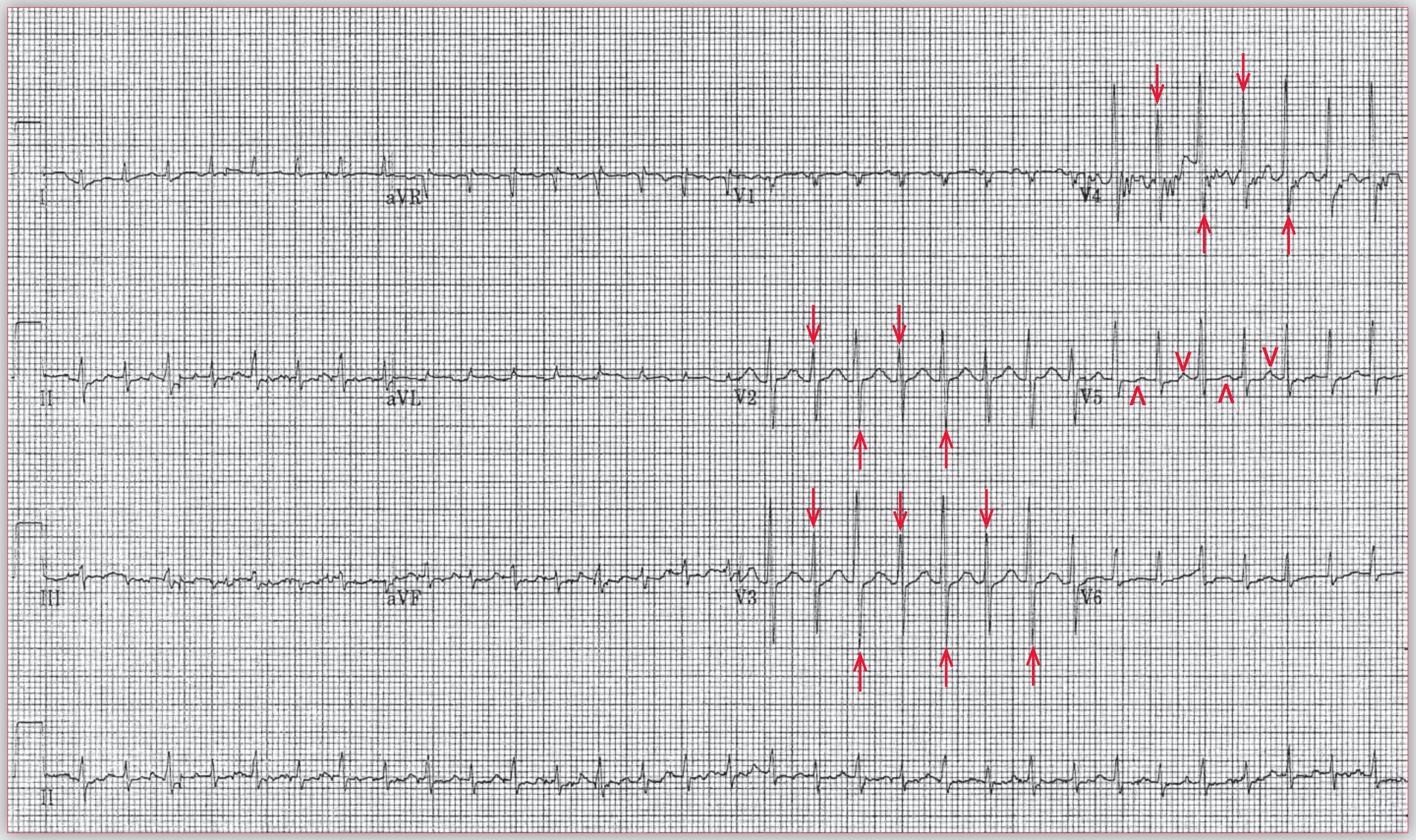
Notes

A 31-year-old man presents to the emergency department with palpitations. He is noted to have a heart rate of 200 bpm. Although his only symptoms are palpitations and the physical examination is otherwise normal, the physician in the emergency department calls cardiology for a stat echocardiogram.

What is the etiology for the tachycardia?

What other abnormalities are noted that prompted the echocardiogram?





ECG 113 Analysis: Atrioventricular nodal reentrant tachycardia (AVNRT), electrical (QRS) and T-wave alternans

There is a regular rhythm with a rate of 200 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complex duration is normal (0.08 sec), and there is a normal axis between 0° and +90° (QRS positive in leads I and aVF). The QT/QTc intervals are normal (240/440 msec). Therefore, this is a narrow complex supraventricular tachycardia without any obvious P waves, *ie*, a no RP tachycardia. The most likely diagnosis is a typical atrioventricular nodal reentrant tachycardia (AVNRT). In this situation, conduction to the ventricles is via the slow pathway and retrograde conduction to the atrial is via the fast pathway. As a result, there is simultaneous atrial and ventricular activation and hence no P wave is seen.

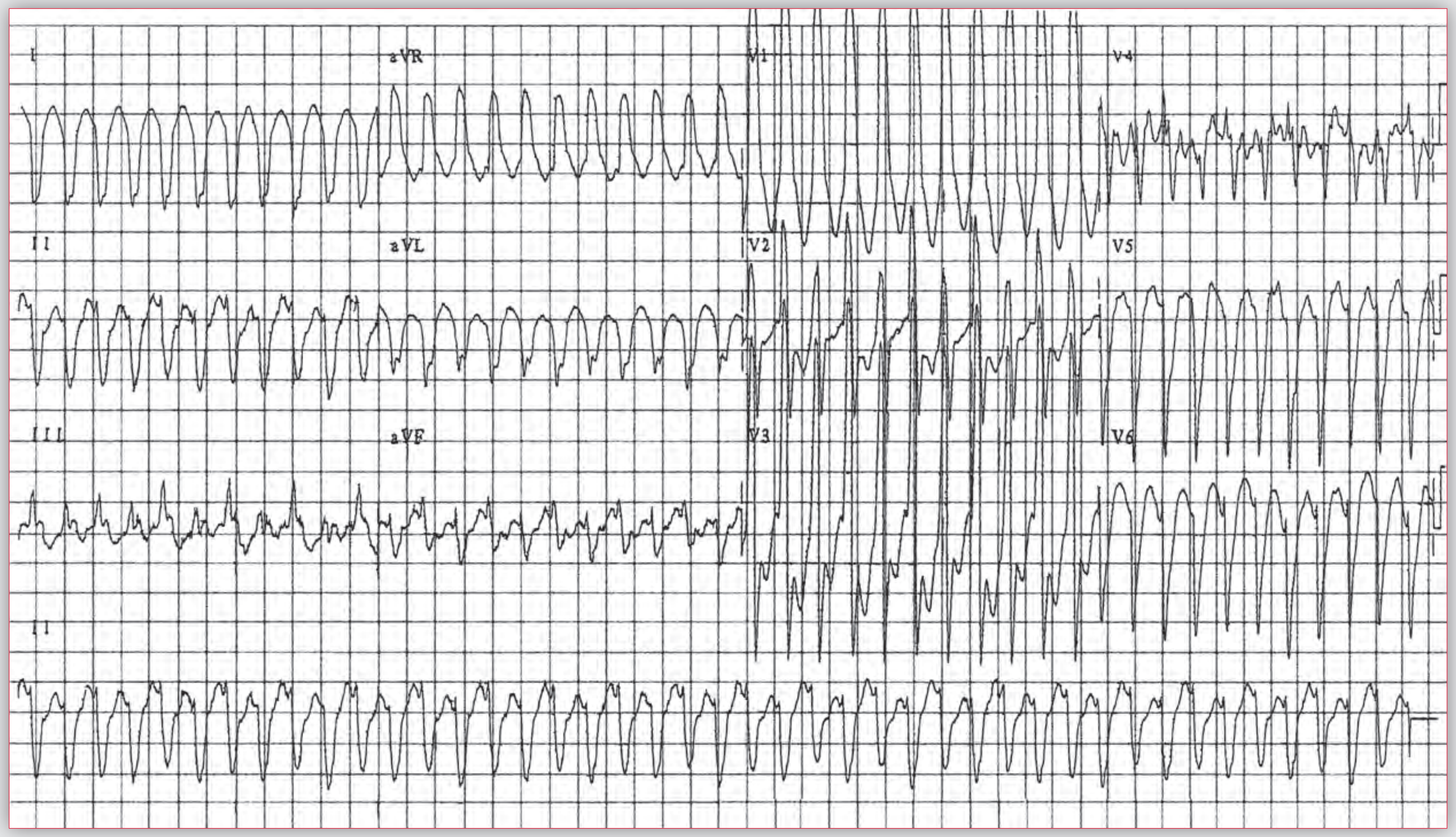
Although the QRS duration is constant, there are beat-to-beat changes in QRS amplitude (↓, ↑). This is termed electrical or QRS alternans. There is also evidence for T-wave alternans (^, v), particularly in lead V5. Electrical alternans in this case is the due to a rapid, regular supraventricular tachycardia and is a result of beat-to-beat changes in calcium influxes. It may occur with any rapid supraventricular tachycardia, including atrial tachycardia, atrial flutter, AVNRT, or atrioventricular reentrant tachycardia (AVRT) as occurs with a preexcitation syndrome. Electrical alternans due to the same mechanism may be seen in a dilated cardiomyopathy, decompensated heart failure or an acute myocardial infarction. It has been reported that it may also be seen with ventricular tachycardia, but this is probably very uncommon. The forms of ventricular tachycardia in which it may be seen are a fascicular tachycardia or a right ventricular outflow tachycardia.

It is also seen with cardiac tamponade as the result of the heart swinging with each beat in a fluid-filled pericardial sac (pendulum effect). In this situation, there is QRS and T-wave alternans present, and there may also be P-wave alternans. It is because of a concern for tamponade that the emergency department physician asked for a stat echocardiogram. ■

Core Case 114

A 31-year-old man with known congenital heart disease, for which he underwent surgery as a child, presents to the emergency department with palpitations that developed while he was jogging. He reports occasional episodes in the past, but they have usually been short-lived.

ECG 114A



Core Case 114

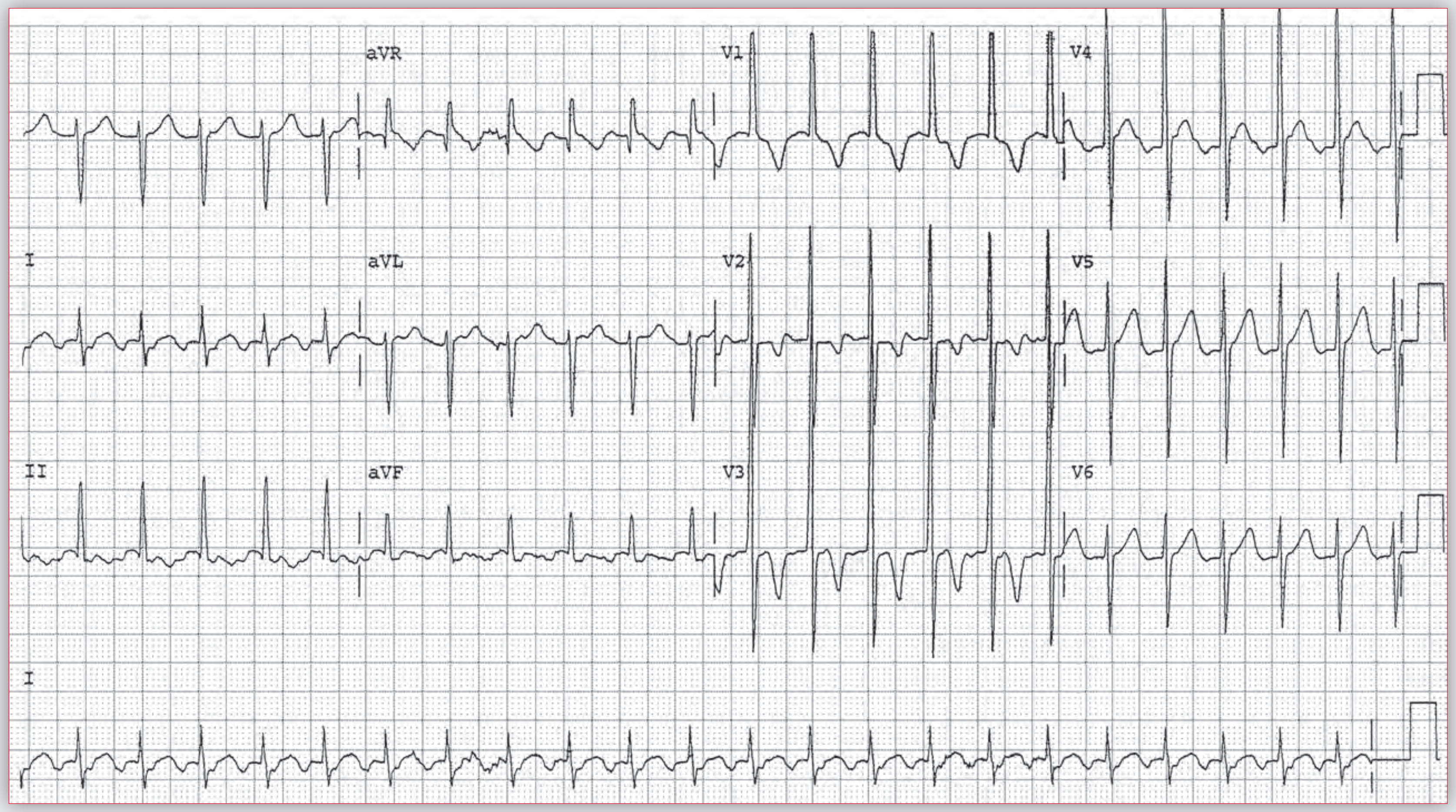
This episode has lasted for 2 hours and as a result he sought medical care. He is noted to have a heart rate of 270 bpm. The rest of his physical examination is unremarkable. Several minutes after presentation, the heart rate abruptly slowed to 140 bpm, and an ECG was obtained (ECG 114B).

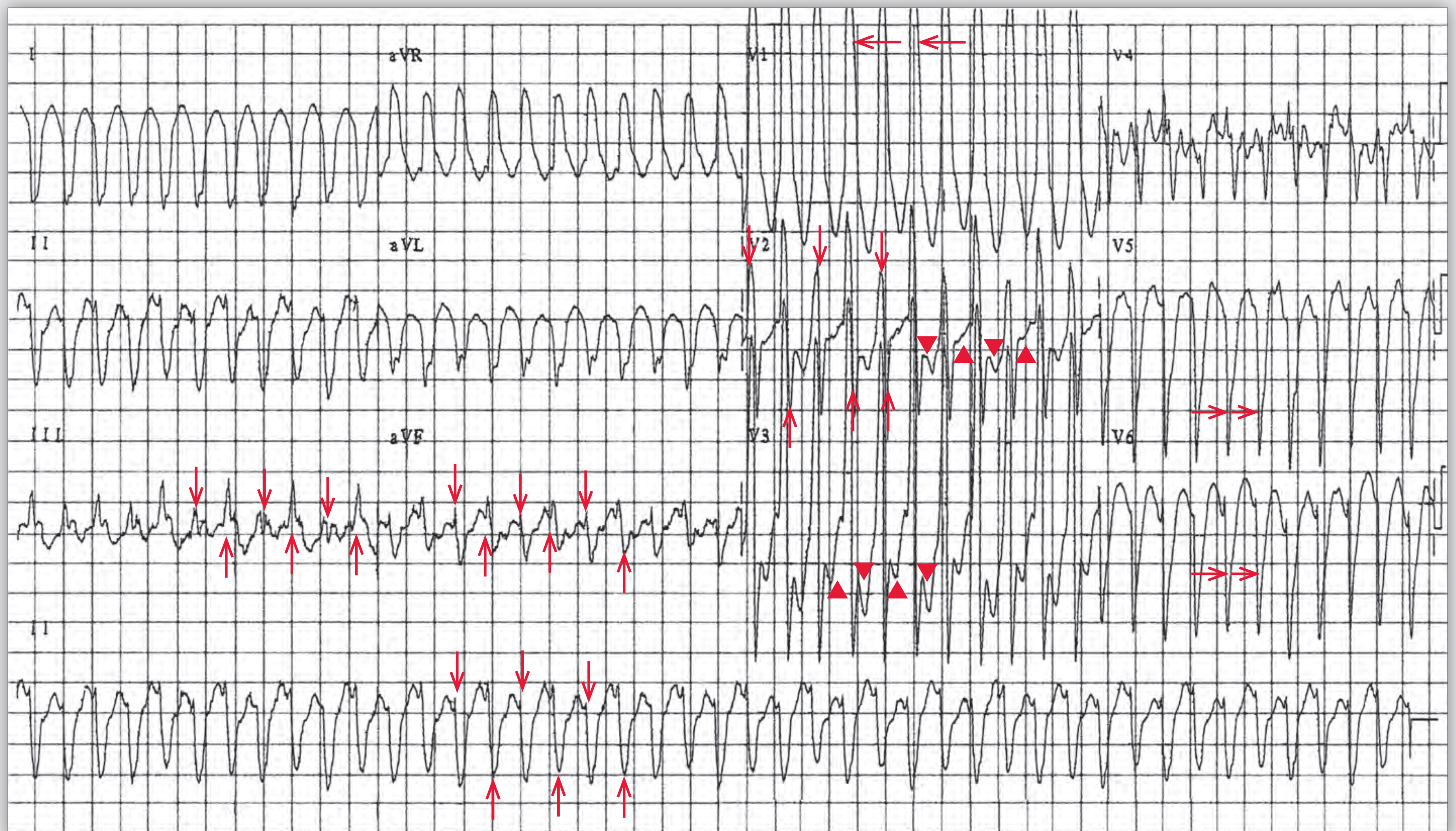
What is the etiology for the tachycardia?

What other abnormalities are noted?

What is the mechanism for the arrhythmia in ECG 114B?

ECG 114B



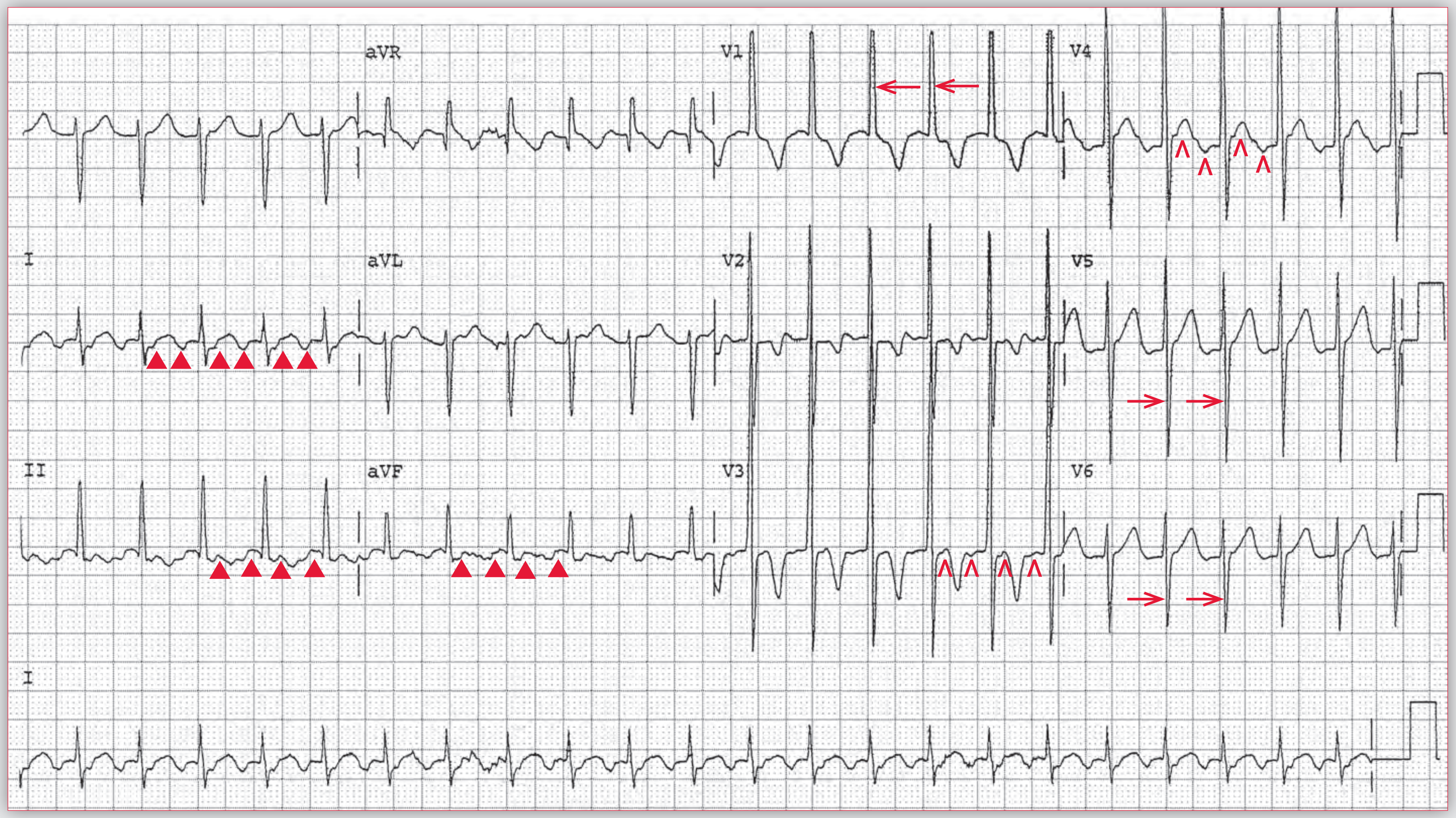


ECG 114A Analysis: Atrial flutter with 1:1 AV conduction, electrical (QRS), and T-wave alternans

ECG 114A shows there is a regular rhythm with a rate of 280 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complex duration is prolonged (0.12 sec), and there is a rightward axis between +90° and +180° (QRS negative in lead I and positive in lead aVF). The QRS complex morphology is unusual with a tall R wave in lead V1 (←), but a small R wave and deep S waves in leads V5–V6 (→). While the morphology resembles a right bundle branch block (RBBB), it is not typical. Hence this is a wide QRS complex tachycardia. There is no obvious atrial activity. The two potential rhythms are atrial flutter with 1:1 conduction or ventricular tachycardia, which at this rate is often referred to as ventricular flutter. The fact that the patient has known congenital heart disease does not help establish a diagnosis. However, there are beat-to-beat changes in QRS amplitude (↓, ↑). This is termed electrical or QRS alternans. There are also beat-to-beat changes in the T waves (▲, ▼) that are T-wave alternans. The presence of electrical (or QRS) and T-wave alternans is more suggestive that this is a supraventricular tachyarrhythmia, and likely flutter with 1:1 AV conduction. Although it has been reported that electrical alternans may occur in ventricular tachycardia, this is very unusual. It may be more frequently seen in a right ventricular outflow tachycardia or a fascicular tachycardia, which is a type of ventricular tachycardia in which one of the fascicles (most often the left posterior fascicle) is part of the circuit of the tachycardia. The QRS complex morphology is not typical for a right ventricular outflow tachycardia (which has a left bundle branch block morphology and a normal axis). Fascicular tachycardias are not usually this rapid, although the right axis and RBBB morphology does suggest a possible left anterior fascicular tachycardia, which is far less common. Therefore, the most likely diagnosis is atrial flutter, with an unusual QRS complex morphology as the result of underlying congenital heart disease. The morphology is suggestive of right ventricular hypertrophy.

QRS and T-wave alternans in atrial flutter with a rapid ventricular rate is a result of beat-to-beat changes in calcium influxes into the ventricular myocardium. It may occur with any rapid supraventricular tachycardia, including atrial tachycardia, atrial flutter, atrioventricular nodal reentrant tachycardia (AVNRT), or atrioventricular reentrant tachycardia (AVRT) as occurs with a preexcitation syndrome. Electrical alternans due to the same mechanism may be seen in a dilated cardiomyopathy, decompensated heart failure, or an acute myocardial infarction. It is also seen with cardiac tamponade as the result of the heart swinging with each beat in a fluid-filled pericardial sac (pendulum effect). In this situation, there is QRS and T-wave alternans present, and there may also be P-wave alternans.

continues



ECG 114B Analysis: Atrial flutter with 2:1 AV conduction, right ventricular hypertrophy, right axis

ECG 114B is from the same patient as ECG 114A and was obtained after the ventricular rate slowed. There is a regular rhythm with a rate of 140 bpm. Although there are no distinct P waves, there are typical atrial flutter waves (at a rate of 280 bpm) seen, primarily in leads II, III, and aVF (▲). They may also be seen in leads V3–V4 (^). Hence this rhythm is atrial flutter with 2:1 AV block. The atrial rate is identical to the ventricular rate in ECG 114A, confirming that this was indeed atrial flutter with 1:1 AV conduction.

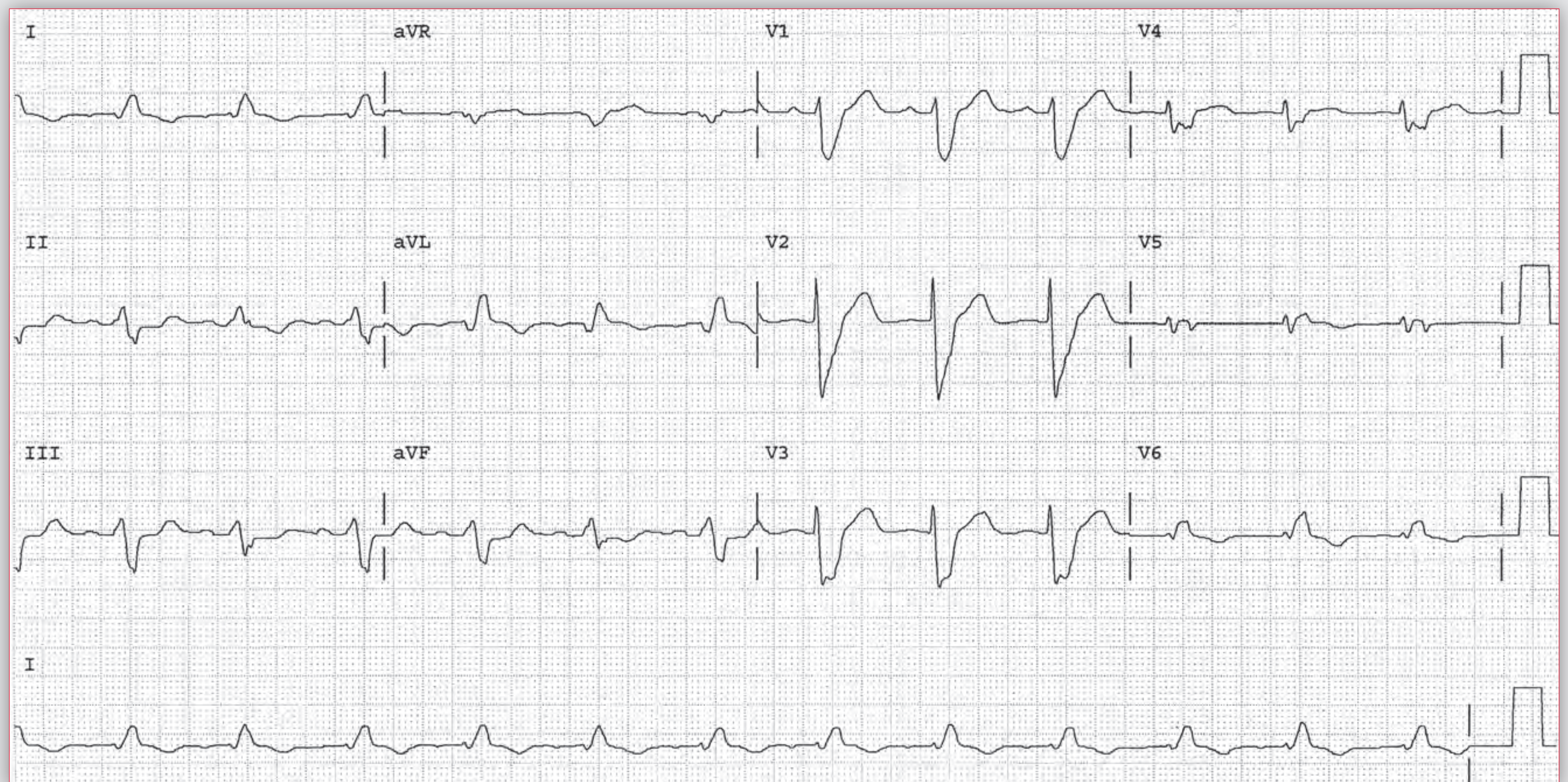
The QRS complex duration is normal (0.08 sec) and there is a rightward axis between +90° and +180° (QRS negative in lead I and positive in lead aVF). The QT/QTc intervals are normal (280/430 bpm). There is a tall R wave in lead V1 (←) and a deep S wave in leads V5–V6 (→), with an S/R ratio > 1. The tall R wave in V1 and the deep S wave in leads V5–V6 are characteristic for right ventricular hypertrophy; this accounts for the rightward axis. Although the nature of the congenital heart disease is not certain, it was most certainly associated with right ventricular volume or pressure load, and most likely due to left to right shunting. ■

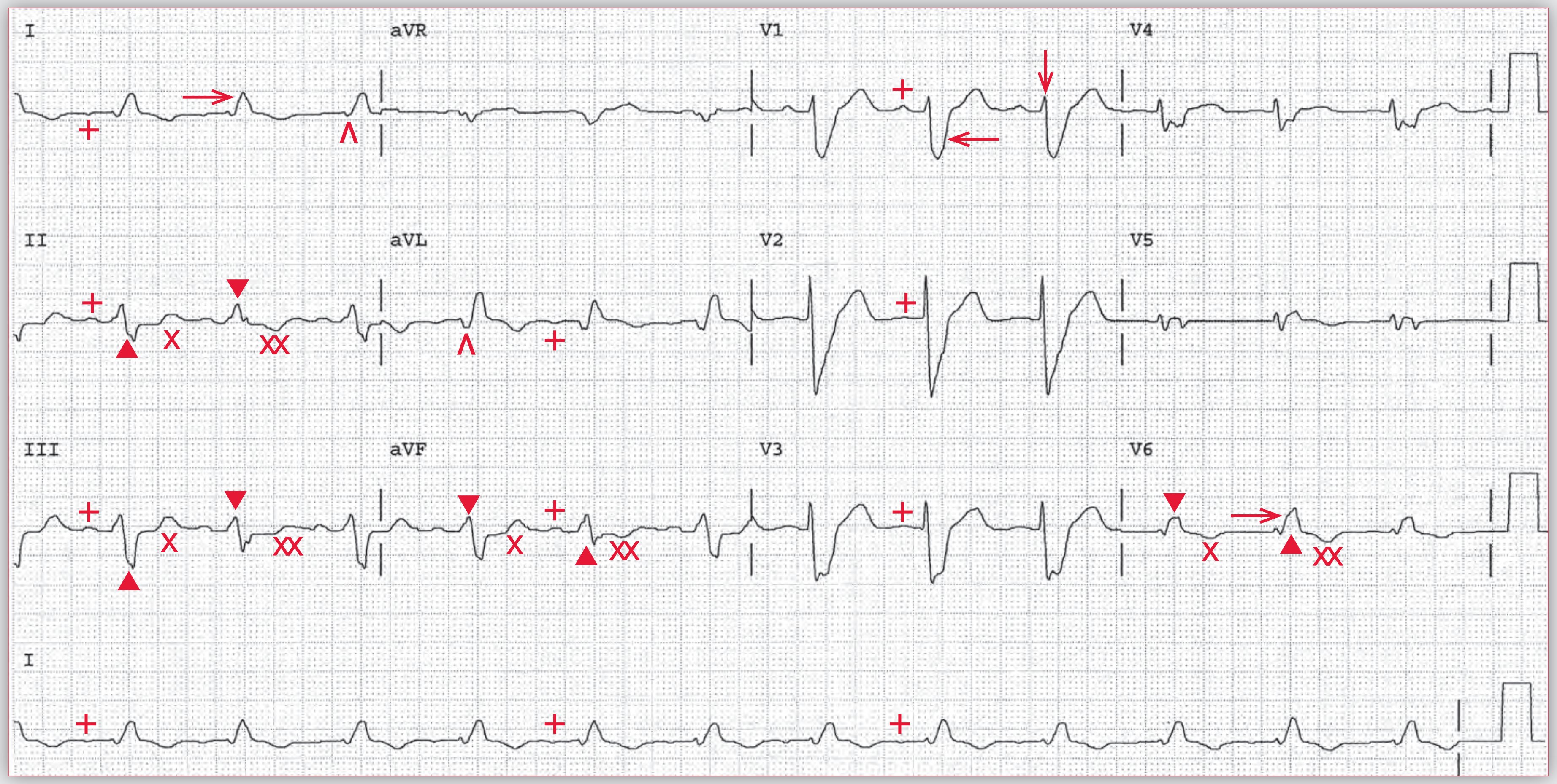
Notes

Core Case 115

A 72-year-old man with advanced cardiomyopathy is awaiting cardiac transplant. He is admitted to the cardiac care unit for inotropic support. His admission ECG is shown.

What are the abnormalities noted on this ECG?





ECG 115 Analysis: Sinus rhythm, marked intraventricular conduction delay, electrical (QRS) alternans, T-wave alternans

There is a regular rhythm with a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads II and aVF, but it is negative in lead I. Therefore, this is an atrial rhythm.

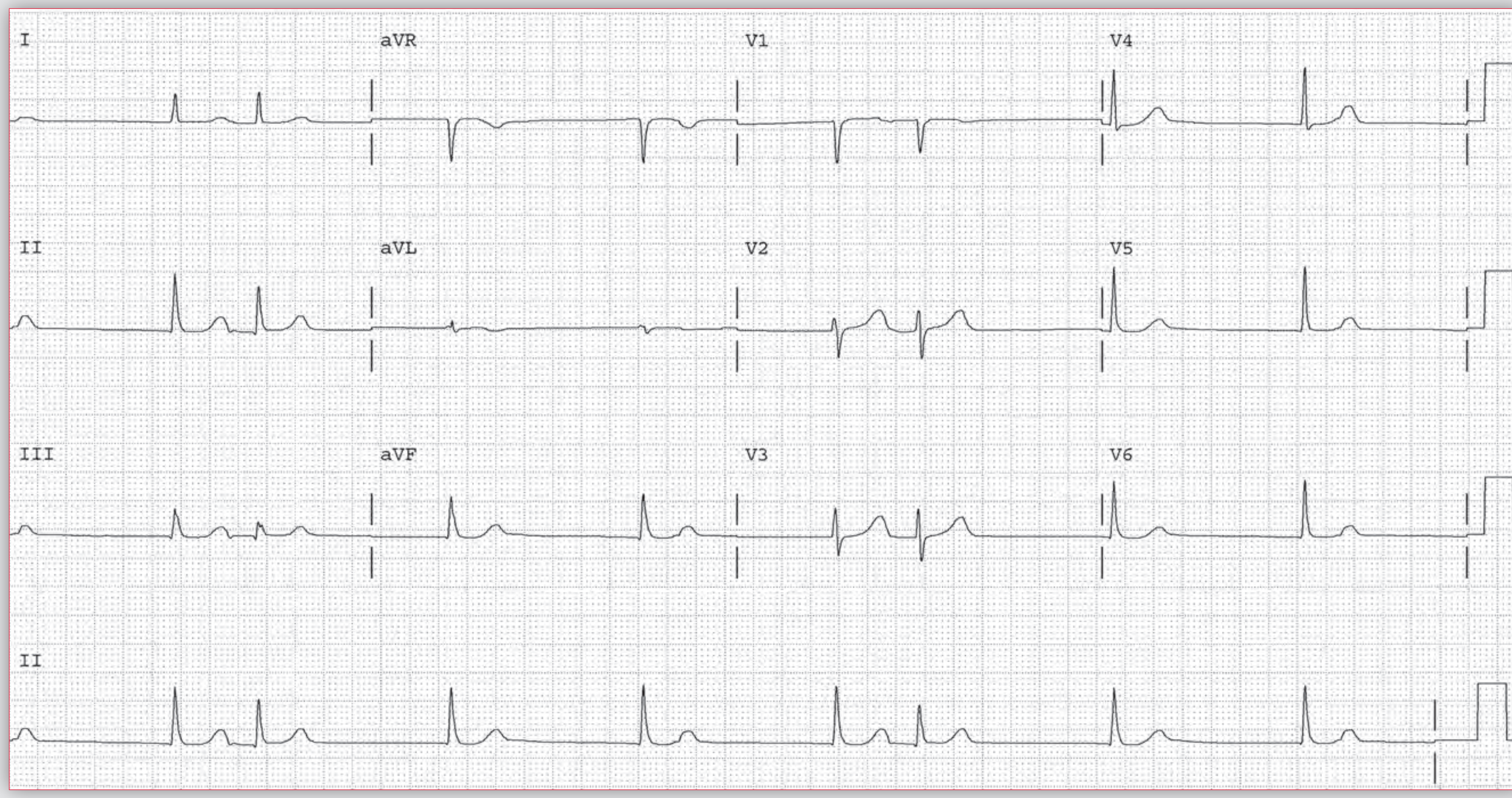
The QRS complex duration is prolonged (0.20 sec). Although the morphology resembles a left bundle branch block (LBBB), with a broad R wave in leads I and V6 (→) and a deep S wave in lead V1 (←), there are Q waves in leads I and aVL (^) and an initial R wave in lead V1 (↓). There cannot be septal forces in LBBB (*ie*, septal Q waves in leads I, aVL, and V5–V6 or septal R wave in lead V1) as the left bundle splits into a left anterior and left posterior fascicle as well as a septal branch that innervates the intraventricular septum, which is the first part of the left ventricle to be activated. With a complete LBBB, there is no impulse conduction through any of these fascicles, including the septal branch. Hence the wide QRS complex is the result of an intraventricular conduction delay. The QRS complex is very wide and this is characteristic of an idiopathic dilated cardiomyopathy. Indeed there is a correlation between the left ventricular ejection fraction and the QRS width, *ie*, the wider the QRS complex, the lower is the ejection fraction. The QT/QTc intervals are long (480/540 msec) but are normal when the prolonged QRS complex duration is considered (380/430 msec).

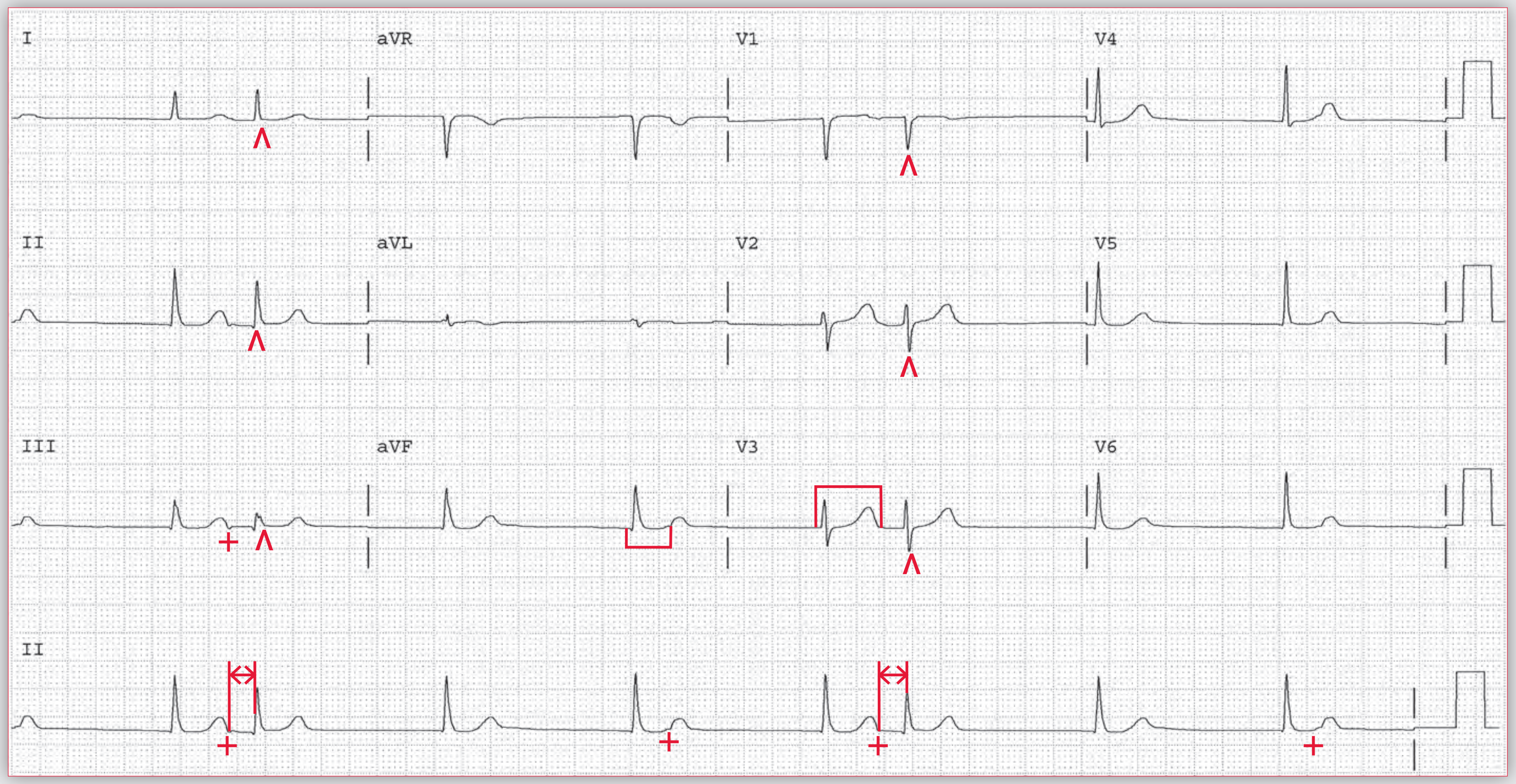
Also consistent with a dilated cardiomyopathy is the presence of beat-to-beat changes in QRS complex (▲, ▼) and T-wave amplitude (x, xx), best seen in leads II, III, aVF, and V6. This is electrical alternans that are the result of beat–to–beat changes in calcium fluxes. ■

Notes

A 44-year-old man with a potential diagnosis of sleep apnea is undergoing a sleep study. On an ECG he is noted to have periods of an irregular rhythm during the study.

What does the ECG show?





ECG 116 Analysis: Junctional rhythm, retrograde (VA) Wenckebach, and echo beats

There is a regular rhythm at a rate of 44 bpm with two early complexes (^) (second and sixth). There are no P waves seen before the QRS complexes, although there is a negative P wave (+) seen in the lead II rhythm strip after the first, fourth, fifth, and eighth QRS complexes. As these are negative in leads II and aVF, they are likely retrograde. Hence there is an underlying junctional rhythm with retrograde P waves. It can be noted that the RP interval is getting longer, *ie*, there is no P wave after the third QRS complex, a short RP interval (⊔) (0.24 sec) after the fourth and a longer RP interval (⊓) (0.40 sec) after the fifth. This is retrograde (VA) Wenckebach. As there is a stable junctional rhythm, with a constant RR interval, the two premature QRS complexes (second and sixth) are responding to the retrograde P wave immediately before them. The PR interval associated with both of these premature complexes is the same (↔) (0.24 sec). All the QRS complexes have a normal duration (0.08 sec) and morphology. There is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/340 msec).

The two premature QRS complexes are called echo beats, *ie*, the retrograde atrial impulse (due to VA conduction) is able to conduct back through the AV node in an antegrade direction, resulting activation of the ventricles. Hence the echo beat will have a supraventricular morphology. Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes or junctional complexes. The VA conduction may occur via an accessory pathway (overt or concealed), one of two AV nodal pathways (dual AV nodal pathways) or through a single AV nodal pathway. With appropriate timing, there may be retrograde conduction through the AV node resulting in atrial activation which is then conducted antegradely through the AV node. Regardless of how the impulse travels back to the atrium, the antegrade ventricular activation is via the normal AV node–His-Purkinje system as the QRS is narrow and resembles the a sinus QRS complex. In this case, the echo beat only occurs at a critical RP interval. The fact that there is retrograde or VA Wenckebach before the echo beat occurs means that retrograde or VA conduction must occur via the AV node, as bypass tracts do not manifest decrimental conduction, which is the hallmark of AV nodal conduction and is the mechanism for Wenckebach (either antegrade or retrograde).

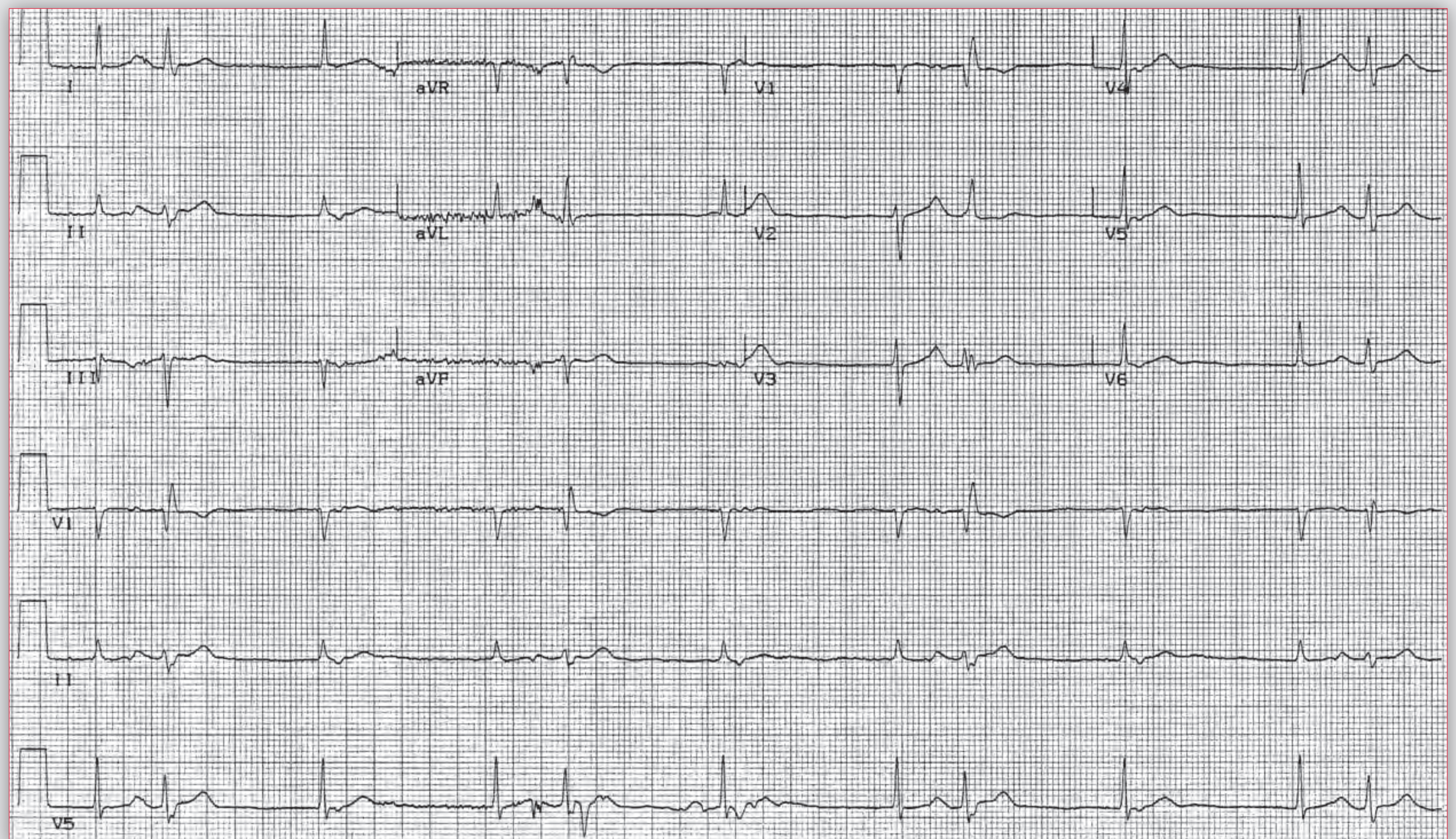
Notes

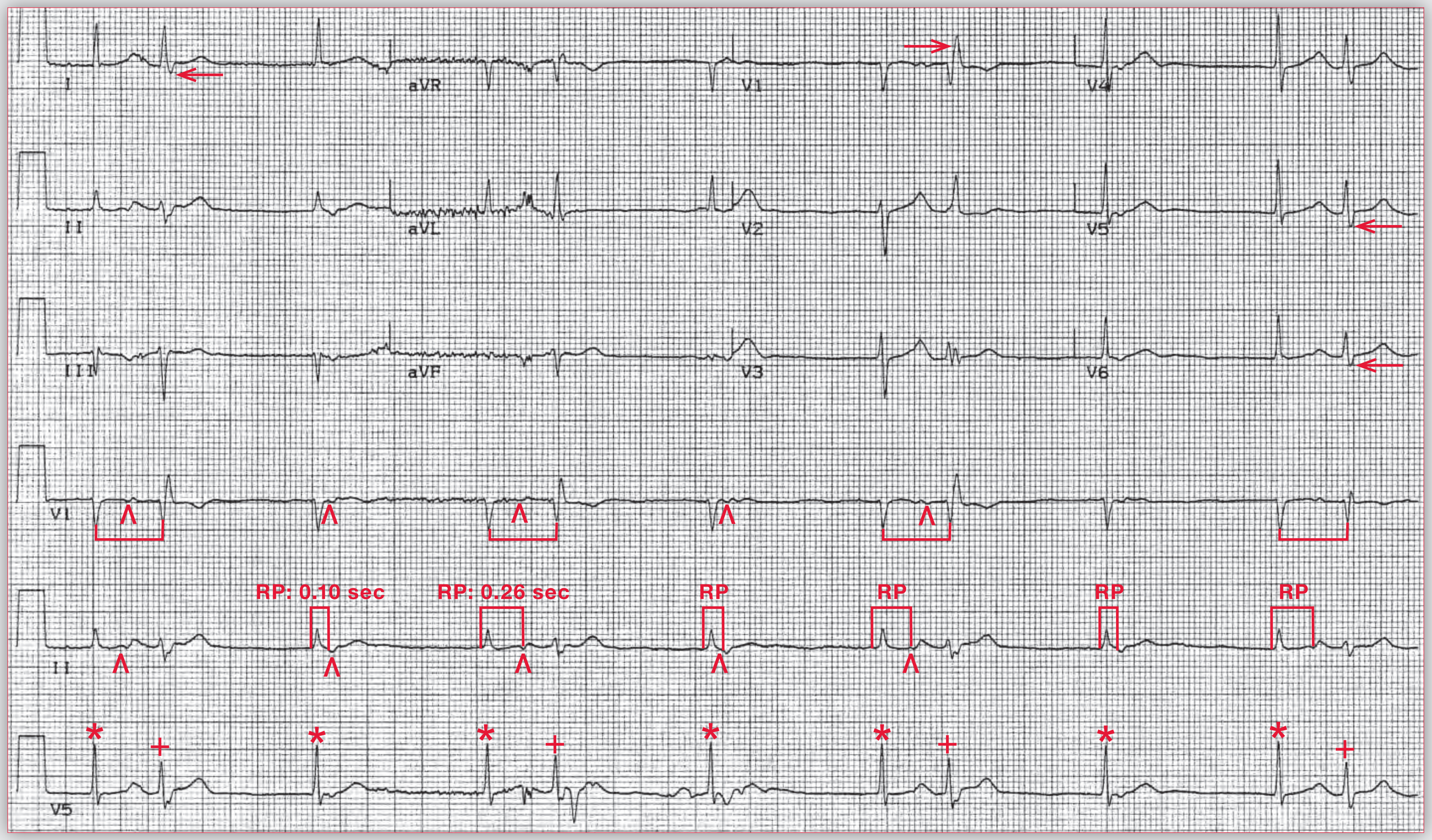
Core Case 117

A 70-year-old man with a history of paroxysmal atrial fibrillation who is receiving therapy with a β-blocker and verapamil presents to the emergency department for complaints of palpitations associated with shortness of breath. He notes that these are the symptoms he feels with the atrial fibrillation. By the time he is seen, the symptoms have resolved. His physical examination is normal. Routine laboratory tests are normal. An ECG is obtained and there is a concern because of an irregularity that was noted.

What is the underlying rhythm?

What is the explanation for the irregular rhythm observed in this ECG?





ECG 117 Analysis: Junctional rhythm with retrograde (VA) Wenckebach and echo beat, rate-related right bundle branch block

There is a regular rhythm present with occasional complexes that are early (+), *ie*, the second, fifth, and eighth, and eleventh. Hence the rhythm would be termed regularly irregular. The early QRS complexes have a fixed coupling interval or fixed relationship (⊔) with the QRS complex that precedes them.

The regular QRS complexes (first, third, fourth, sixth, seventh, ninth, and tenth) (*) have a normal duration (0.08 sec) and a leftward axis between 0° and −30° (positive QRS complex in leads I and II and negative in aVF). They have a normal morphology. There are no P waves in front of any of these complexes; these are junctional complexes and the underlying rhythm is junctional. The RR interval between the third and fourth, sixth and seventh, and ninth and tenth is the same (*ie*, rate of 48 bpm). The QT/QTc intervals are normal (460/410 msec). P wave (^) can be seen after each of these junctional complexes. The P waves are negative in leads II, aVF, and V4–V6; hence they are retrograde P waves due to VA conduction from the junctional complex. However, there are two different RP intervals (*ie*, interval between the QRS complex and the retrograde P wave) (⊓). The RP interval after QRS complexes 3, 6, and 9 is very short (0.10 sec) and the same each time; the retrograde P wave (^) is the negative wave immediately after the QRS complex. The RP interval after the first, fourth, seventh, and tenth QRS complexes is longer (0.26 sec) and is the same each time. Hence there is evidence of retrograde or VA Wenckebach. It is also possible that the retrograde or VA conduction is occurring via two different AV nodal pathways, *ie*, a slow and fast pathway. After the P wave with the longer RP interval, there is an early QRS complex (+), with a constant relationship (*ie*, there is fixed coupling) to the retrograde P wave that precedes it (*ie*, constant PR interval, which is 0.20 sec). The early QRS complexes are in response to the negative P wave that precedes them. Hence these are called echo beats, which are a result of VA conduction from the junctional complexes. After retrograde atrial activation there is antegrade conduction through the AV node–His-Purkinje system, resulting in normal ventricular activation. Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes or junctional complexes. While the resulting echo beat is due to conduction through the normal AV node–His-Purkinje system, the pathway for VA conduction may be an overt or concealed bypass tract, one of two dual AV nodal pathways or with the appropriate timing through the same AV nodal pathway. The fact that there are two different RP intervals indicates the presence of either dual AV nodal pathways (slow and fast) or retrograde Wenckebach (due to decrimental conduction), which means that the VA conduction must be through the AV node, as bypass tracts do not exhibit different rates of VA conduction.

The echo beats have a prolonged duration (0.12 sec) and have a typical right bundle branch pattern with an RSR′ in lead V1 (→) and broad terminal S wave in leads I and V4–V6) (←). The right bundle branch block aberration is the result of a shorter RR interval; hence this is a rate-related right bundle branch block.

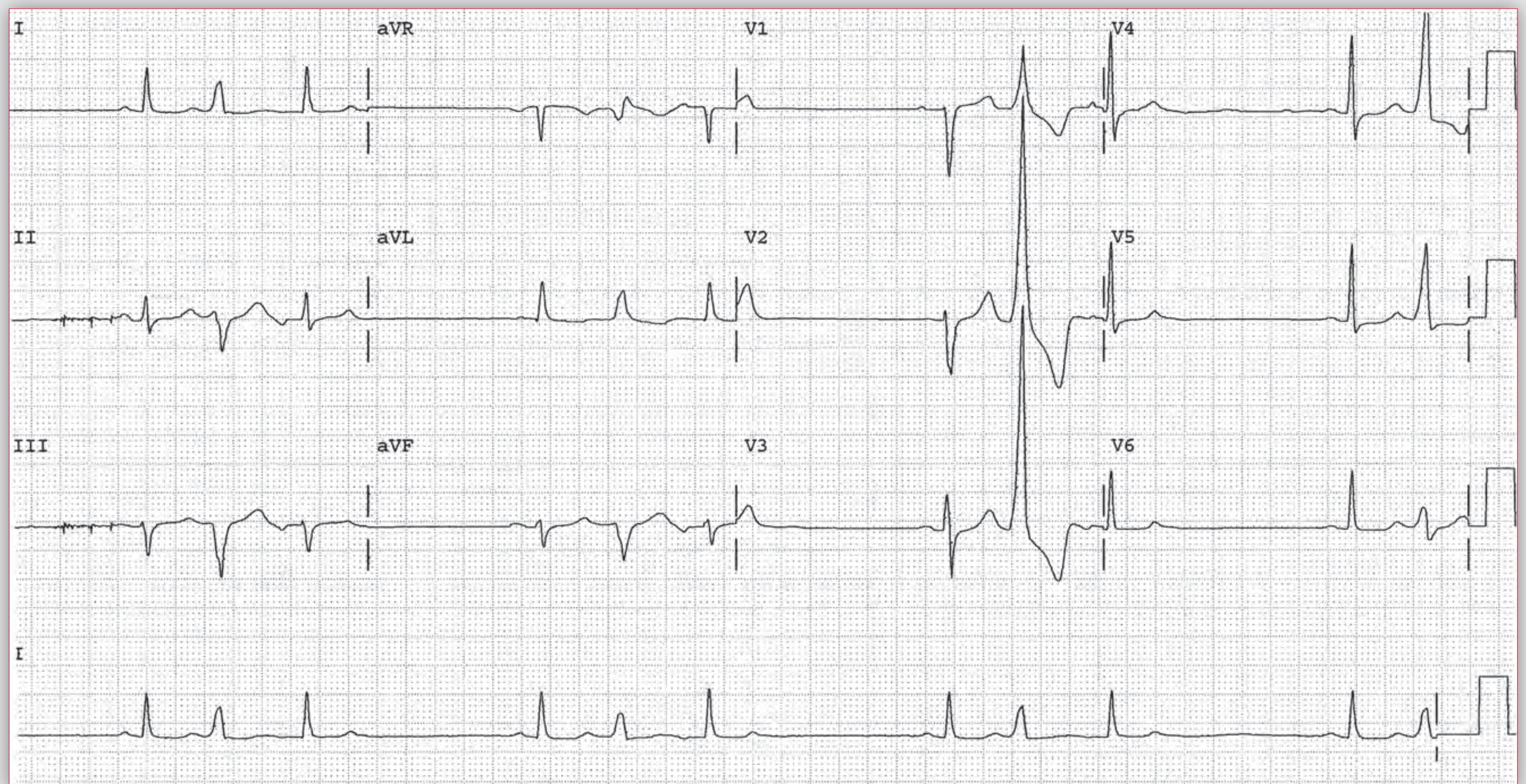
In this case, the junctional rhythm is possibly the result of an episode of atrial fibrillation that abruptly terminated. As a result of a β-blocker and calcium-channel blocker, it is likely that there was suppression of sinus node activity, resulting in a junctional rhythm. ■

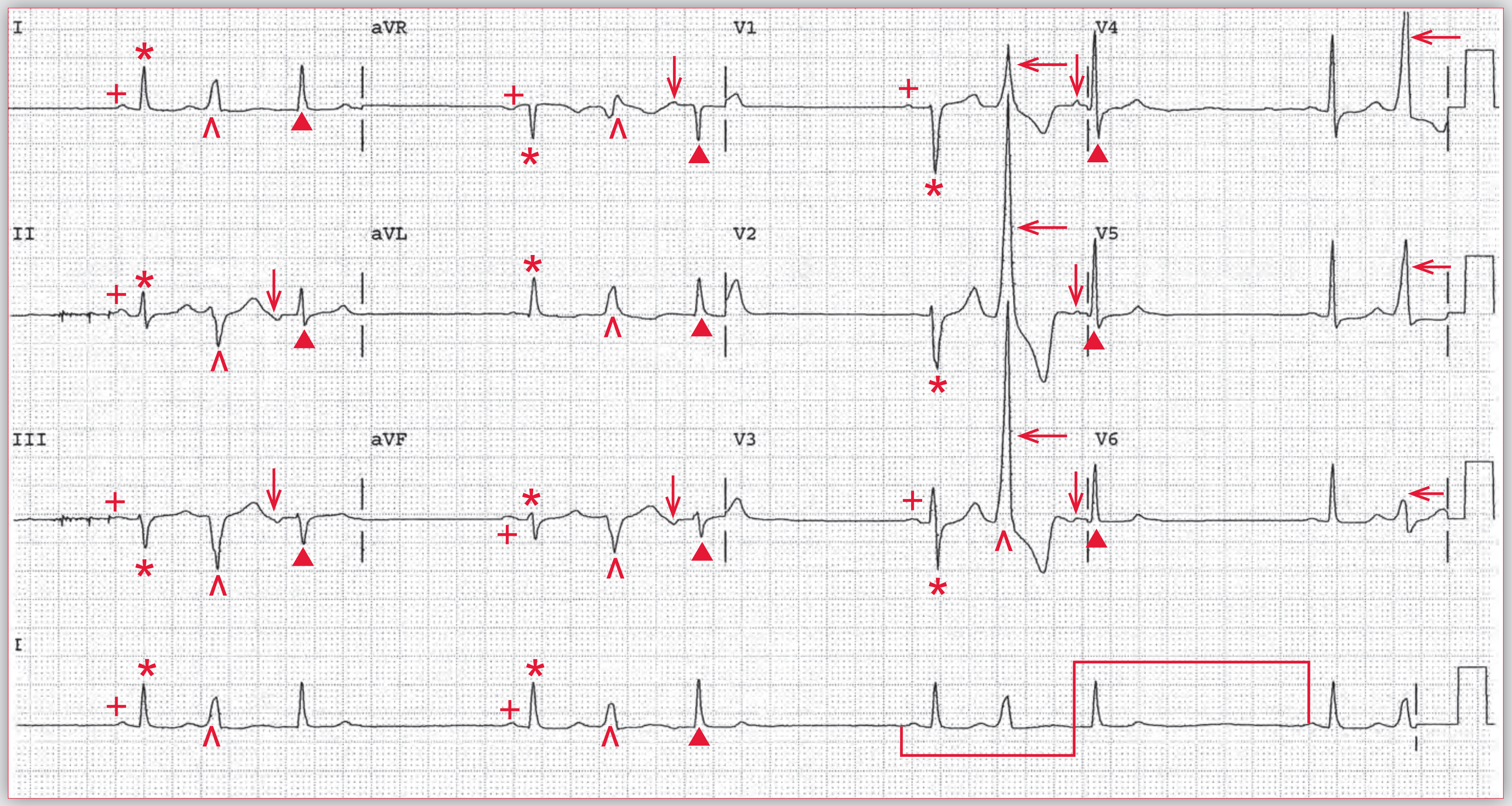
Notes

A 56-year-old woman is seen by her internist for flu-like symptoms that have been present for several days with fever, chills, nasal congestion, and a productive cough. Her physical examination is unremarkable except for an irregular pulse. As a result, an ECG is obtained.

What does it show?

Is there any cause for concern?





ECG 118 Analysis: Normal sinus rhythm with premature ventricular complexes, retrograde (VA) conduction and echo beats, left axis

There is group beating with three QRS complexes that occur in a regularly irregular pattern. The first QRS complex (*) of the group is narrow (0.08 sec), has a leftward axis between 0° and –30° (positive QRS complex in leads I and II and negative in aVF) and has a P wave (+) before it with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence these are sinus complexes. The QT/QTc intervals are normal (400/420 msec).

The second QRS complex (^) has a prolonged duration (0.14 sec) and there is no preceding P wave. Also noted is that these complexes have positive concordance across the precordium (*ie*, tall R wave V1–V6) (←). Positive concordance is seen with QRS complexes that are the result of direct ventricular activation, including paced complexes, preexcitation (primarily Wolff-Parkinson-White) due to an accessory pathway, or ventricular complexes. There are no pacemaker stimuli seen and these complexes are not preceded by atrial activity, which excludes preexcitation, as a preexcitated complex must have initial atrial activity as the accessory pathway begins on the atria. Hence these are premature ventricular complexes.

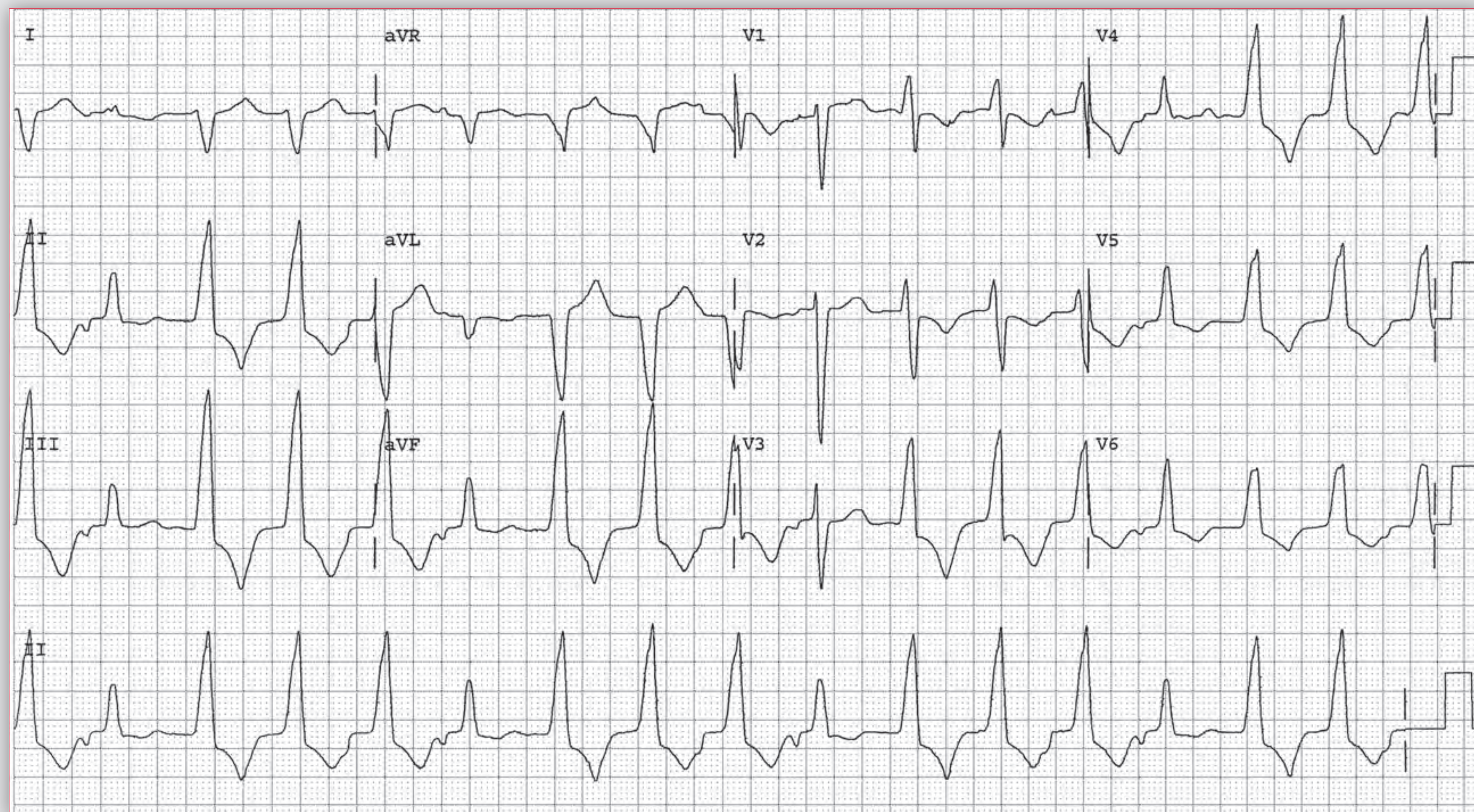
The third QRS complex (▲) that follows the premature ventricular complex is identical to the first QRS complex in width, morphology, and axis. It also has the same QT/QTc intervals. Hence it is supraventricular in origin. There is a P wave (↓) seen before this QRS complex, most apparent in leads II, III, and aVF where it is negative, and also in lead aVR where is it positive. It has a different morphology from the sinus P wave. It should be pointed out that this is not an interpolated premature ventricular complex, *ie*, a complex that is occurring between two sinus QRS complexes and is not associated with a pause and does not alter the sinus PP interval. As can be seen, the PP interval between the sinus P wave and the negative P wave that follows the premature ventricular complex (⊔) is much shorter than the PP interval between the negative P wave and the following sinus P wave (⊓). In addition, the P-wave morphologies of the complex before and the complex after the premature ventricular complex are different. Therefore, the negative P wave may either be a premature atrial complex or be due to retrograde or VA conduction resulting from the premature ventricular complex. It is most likely a retrograde P wave. As a result of the retrograde P wave and retrograde activation of the atrium, the impulse then enters the AV node antegradely and results in a QRS complex that is due to conduction via the normal His-Purkinje system. There is a fixed interval (constant relationship) between the premature ventricular complex and the retrograde P wave (*ie*, RP interval) and also a fixed interval between the retrograde P wave and QRS complex that follows it (*ie*, PR interval).

The third QRS complex that results from retrograde VA conduction is termed an echo beat, *ie*, the retrograde atrial impulse is able to penetrate the AV node in an antegrade direction, resulting in conduction through the His-Purkinje system and a supraventricular complex. Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes, or junctional complexes. ■

Core Case 119

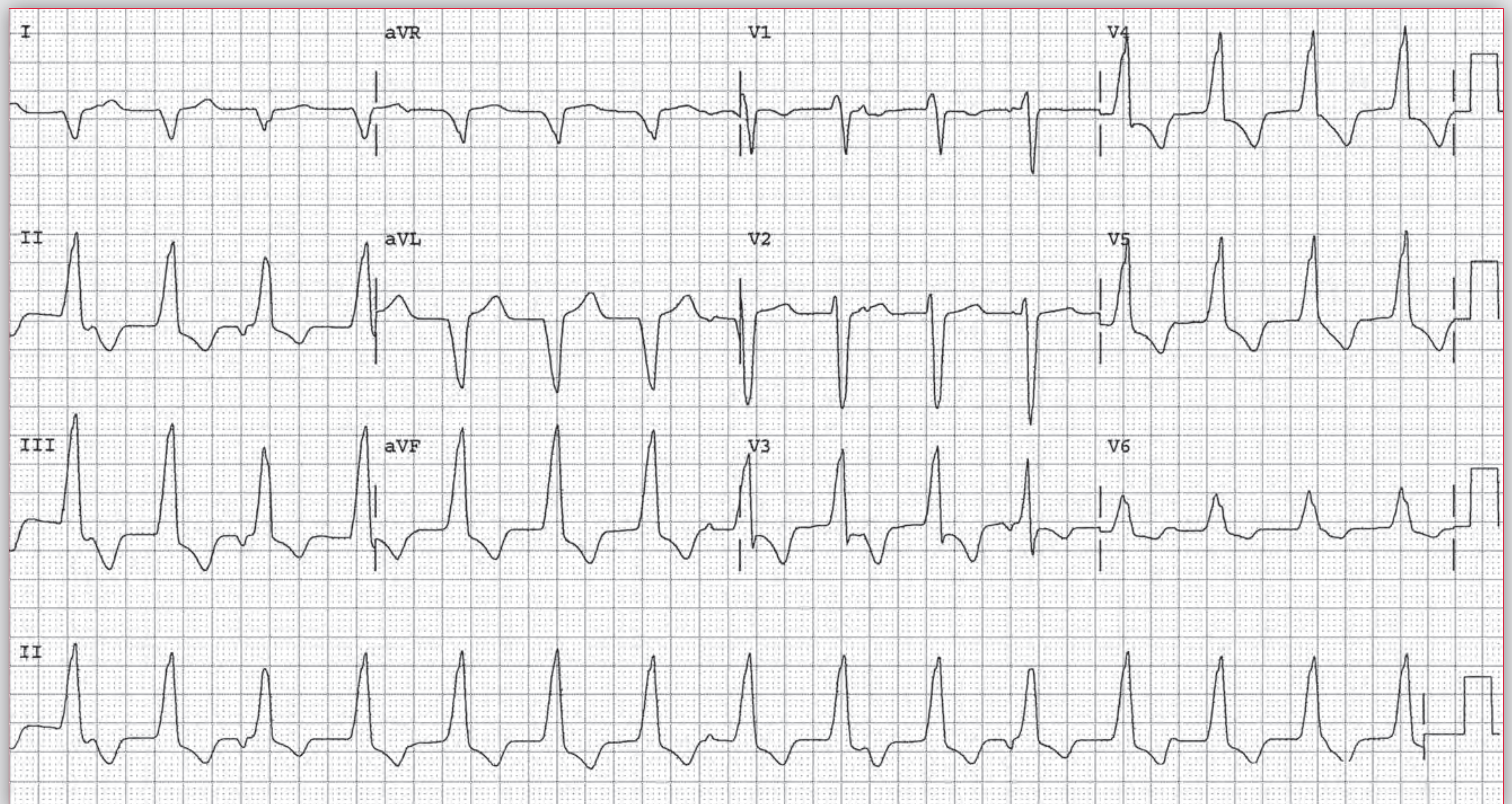
A 77-year-old woman with a known dilated cardiomyopathy presents to the hospital because of lightheadedness, profound fatigue and increased shortness of breath. She states that her pulse rate,

ECG 119A



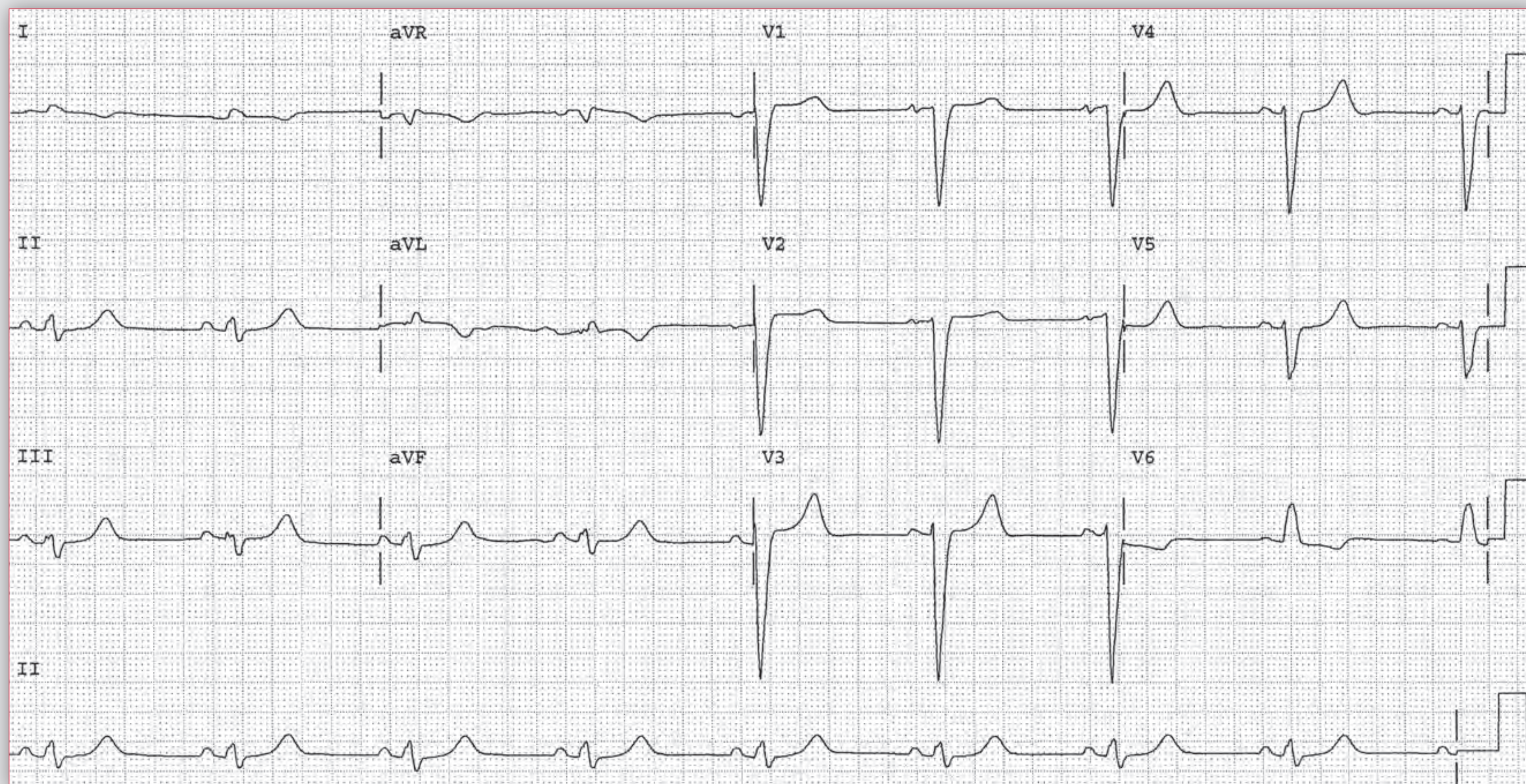
which she monitors frequently, has been rapid. It is usually 50–60 bpm, but for the past 1–2 days, she has noted that it is 100 bpm. An ECG (119A and 119B) is obtained. They are compared to her baseline ECG (ECG 119C).

ECG 119B



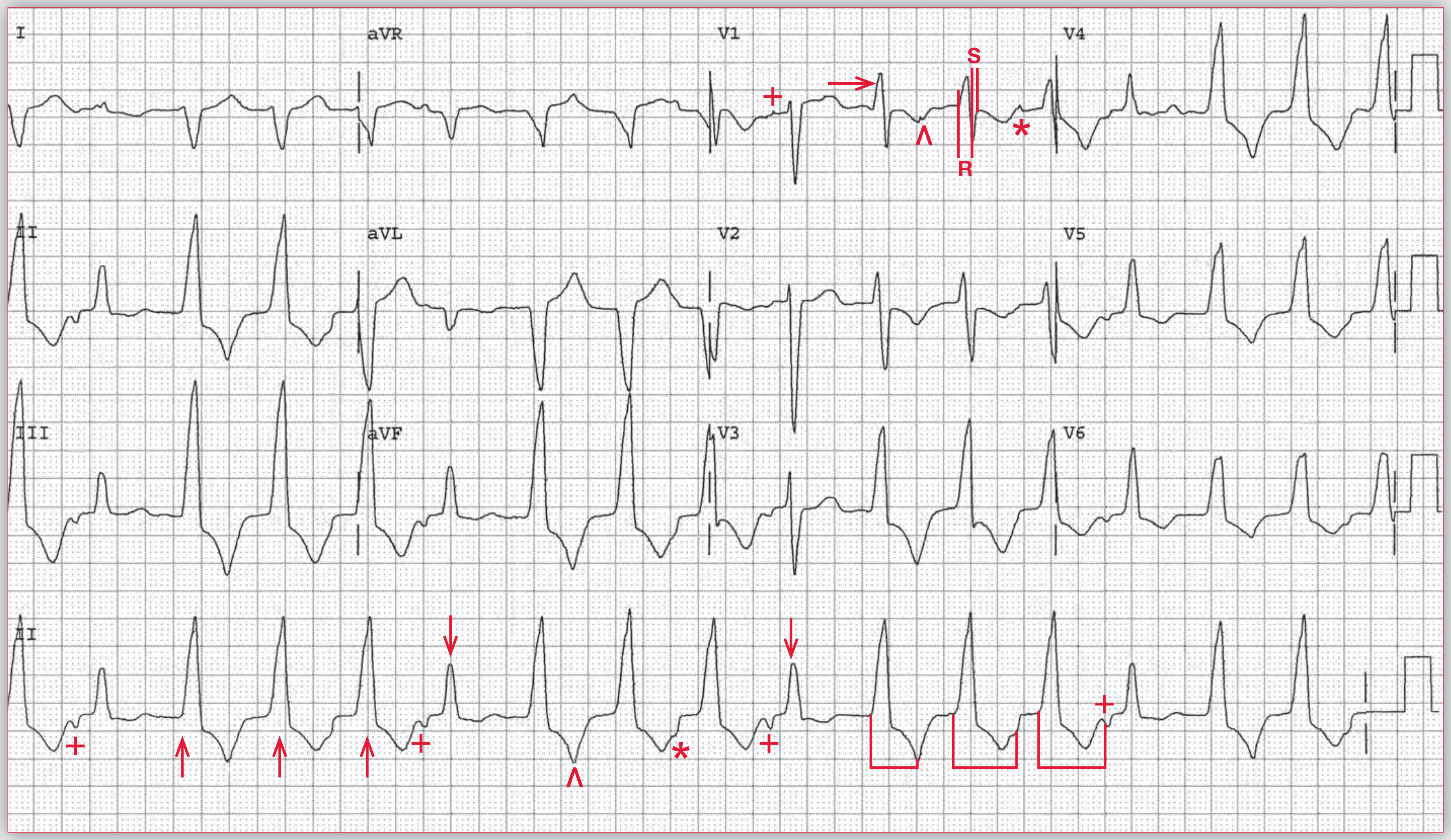
Core Case 119

ECG 119C



What is the rhythm?

What other abnormality is present? For comparison, her baseline ECG (ECG 119C) is presented.



ECG 119A Analysis: Accelerated idioventricular rhythm, retrograde (VA) Wenckebach, echo beats

ECG 119A shows there is a regular rhythm at a rate of 100 bpm. There is variability of the QRS complex morphology and width; there is repeating pattern of group beating consisting of three wider QRS complexes (↑) with a duration of 0.18 sec and one narrower QRS complex (↓) (0.14 sec) that also has a different amplitude. The QRS axis is rightward between +90° and +180° (negative QRS complex in lead I and positive in lead aVF). There is a tall R wave in lead V1 (→) and the R wave is broader than the S wave, a feature associated with a ventricular complex. In addition, the QRS complexes do not have either a right or left bundle branch block morphology. Therefore, this is an accelerated idioventricular rhythm, also called slow ventricular tachycardia. The QT/QTc intervals are prolonged (400/515 msec) but are only slightly prolonged when the prolonged QRS complex duration is considered (360/460 msec).

There are no P wave seen before any of the wider QRS complexes, but there is a P wave (+) after the third wider QRS complex and before the narrower QRS complex (↓). The P wave is negative in leads I, II, aVF, and V4–V6. Therefore, it is not originating from the sinus node. Negative P waves are either retrograde due to VA conduction or a low atrial focus. As this is a ventricular rhythm, this is most likely a retrograde P wave due to VA conduction. Although there are no other obvious P waves seen associated with the wider QRS complexes, noted are changes in the T waves in the lead II rhythm strip that usually indicate superimposed P waves. The first wider QRS complex in the lead II rhythm strip has a deep and narrow negative waveform (^), and the second wider QRS complex has a negative notching on the upstroke of the T wave (*). These P waves also appear to be negative. The P waves are best seen in lead V1. As they are seen with the wider QRS complexes, they are not premature atrial waves, but rather they are associated with the QRS complex. Hence this is a ventricular rhythm with retrograde P waves as a result of VA conduction. However, it can be noted that there is a progressive lengthening of the RP interval (⊔) from 0.32 to 0.40 to 0.48 sec, *ie*, retrograde Wenckebach. This occurs in a repeating pattern. The retrograde P wave after the third wide QRS complex (+) results in the narrower QRS complex (↓) that is difference in morphology from the other three QRS complexes. Indeed, it has a more normal looking morphology in the precordial leads with normal R wave progression. It is in response to the retrograde P wave and is an echo beat.

Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes or junctional complexes. As a result of the retrograde P wave, there is antegrade impulse transmission through the normal AV node–His-Purkinje system, *ie*, the retrograde P wave echoes back to the ventricle. The echo beat will be identical to the supraventricular QRS complex. A retrograde impulse can travel back to the atrium via an overt or concealed bypass tract, one of two dual AV nodal pathways, or back and forth through a single AV nodal tract. The fact that there is retrograde Wenckebach means that the retrograde conduction must be via the AV node as this structure demonstrates decrimental conduction, the mechanism for Wenckebach (antegrade or retrograde). Bypass tracts do not demonstrate decrimental conduction.

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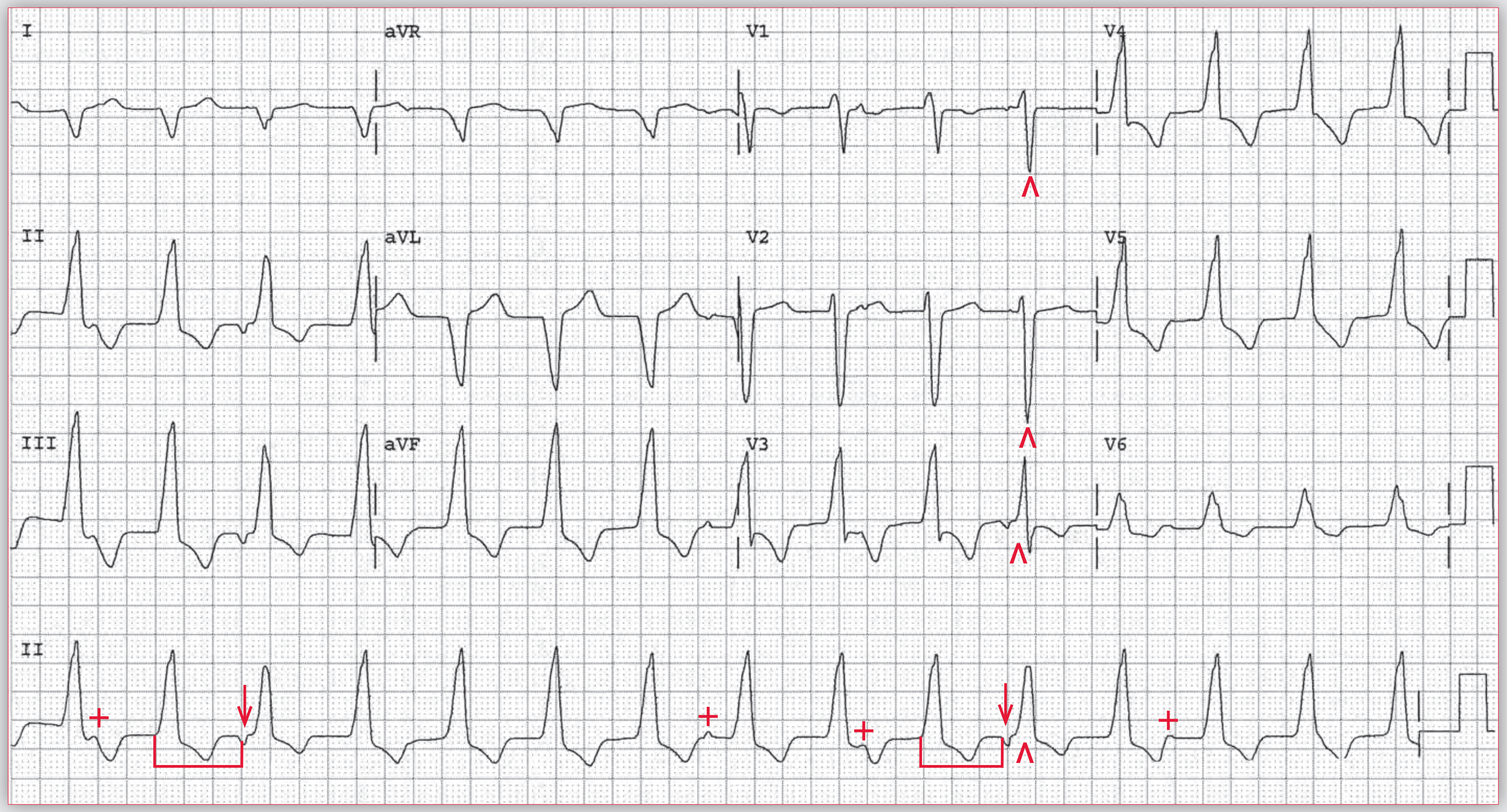

ECG 119B Analysis: Accelerated idioventricular rhythm, AV dissociation, echo beats

ECG 119B is from the same patient as ECG 119A. There is a regular rhythm at a rate of 96 bpm. The QRS complex morphology, duration (0.18 sec) and axis are the same as those in ECG 119A. The QT/QTc intervals are also the same. Therefore, this is an accelerated idioventricular rhythm, the same as the rhythm in ECG 119A. There are P waves (+) noted after QRS complex 1, before complex 8, after complex 9 and after complex 12. They are positive in leads II and aVF and are sinus in origin. The PR interval is not constant and there is no relation between these P waves and the QRS complex; this is AV dissociation. As the ventricular rate is faster than the atrial rate, this is an accelerated lower focus. As the QRS complexes are wide, without any specific bundle pattern, the rhythm is ventricular. However, there are also P waves that are negative in lead II (before complexes 3 and 11 (↓). There is a fixed RP (0.56 sec) (⊔) and PR interval (0.14 sec). Similar to the negative P waves seen in ECG 119A, these are retrograde due to VA conduction from the ventricular complex. Following these retrograde P waves are QRS complexes (^) that are slightly narrower (0.14 sec) and have a morphology and axis that are similar to the narrower QRS complexes in ECG 119A (particularly in leads V1–V2) although they are slightly wider, yet they also have a similarity to the ventricular complexes (particularly in leads I, II, and III). Hence these are echo beats, but they are fused with the ventricular QRS complexes, *ie*, there is activation via the normal AV node–His-Purkinje system (*ie*, echo) that is simultaneous with the occurrence of the ventricular complex. Therefore, this ECG demonstrates both AV dissociation as well as retrograde P waves with echo beats.

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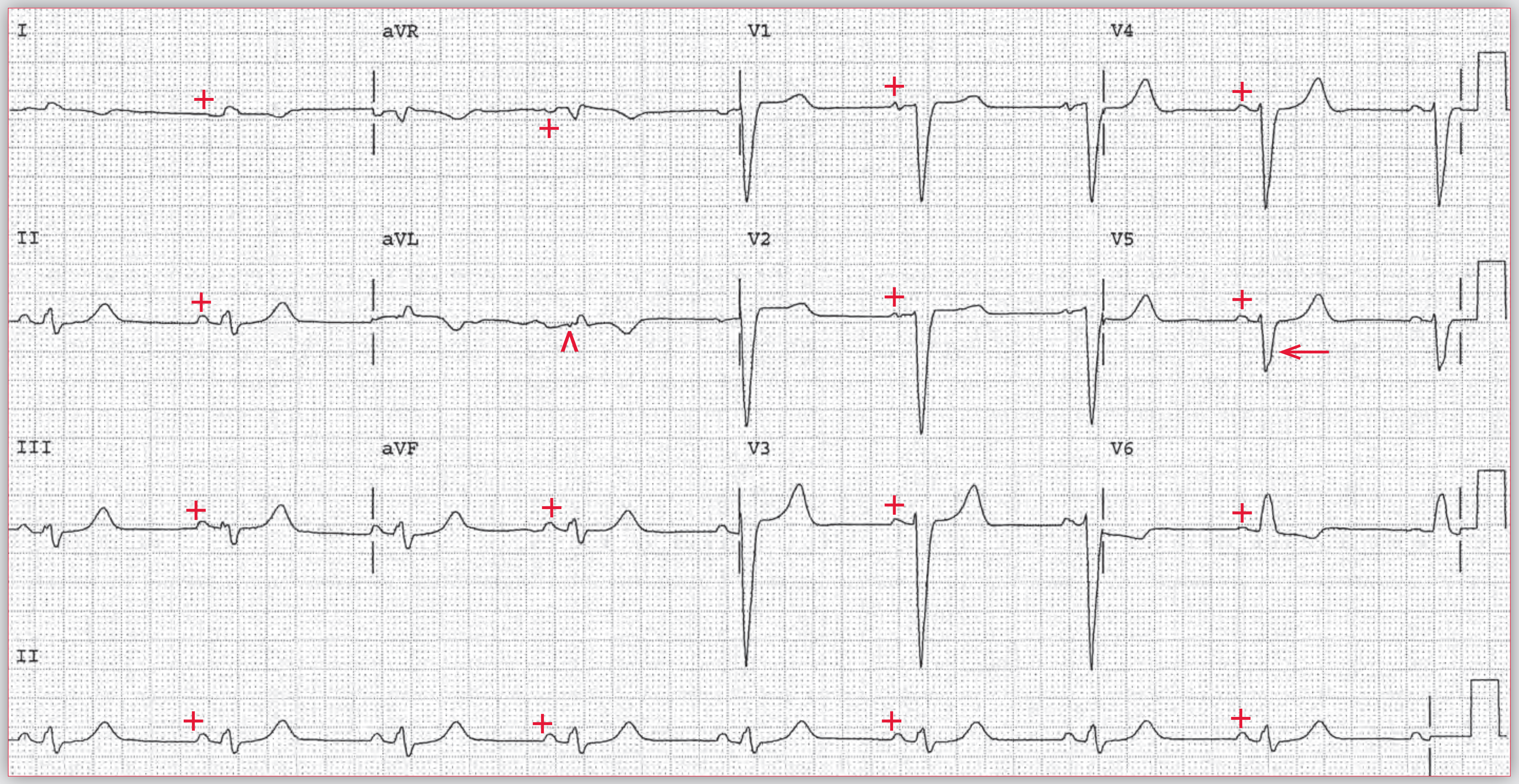


ECG 119C Analysis: Sinus bradycardia, intraventricular conduction delay, left axis, low voltage limb leads

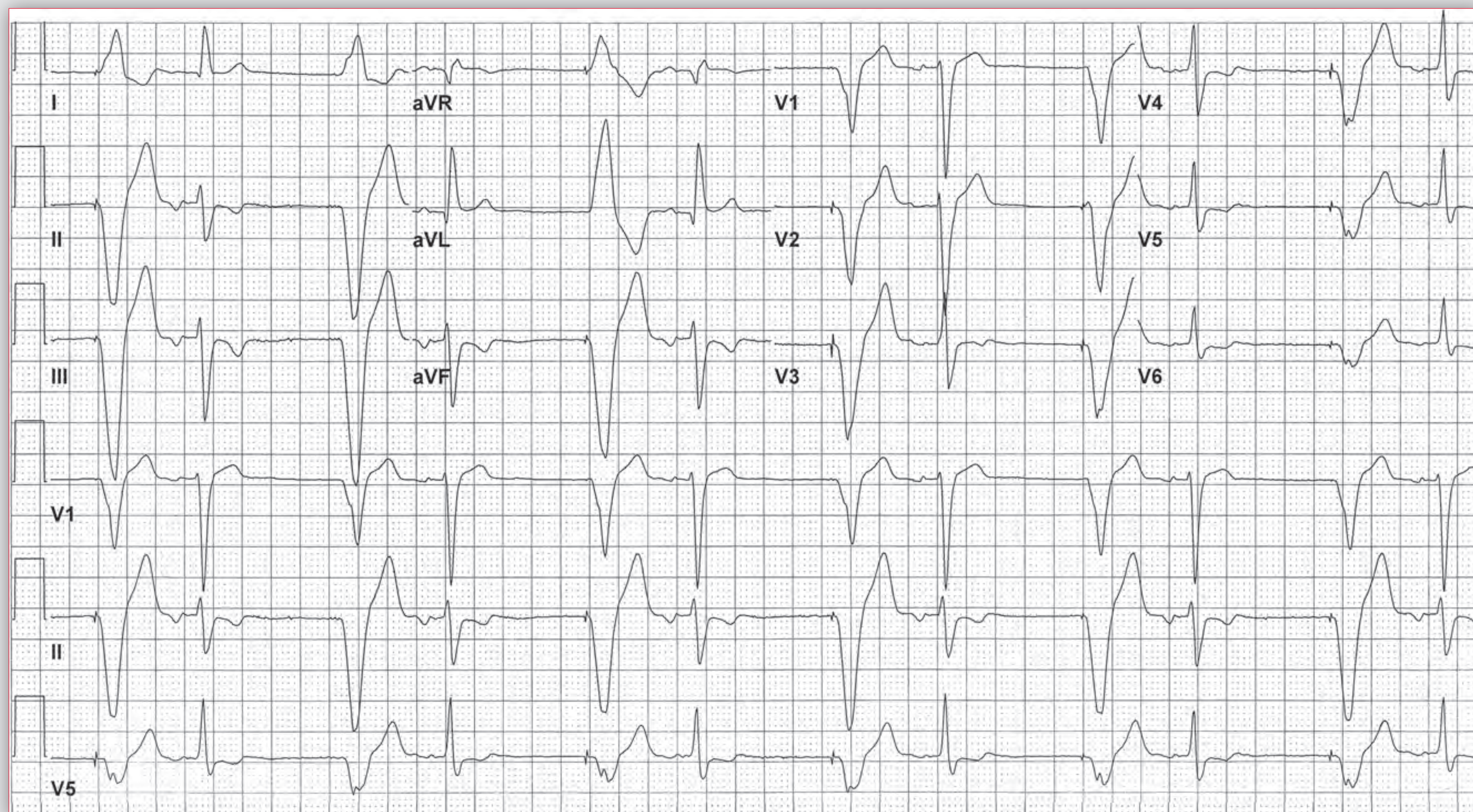
ECG 119C shows there is a regular rhythm at a rate of 50 bpm. There are P waves (+) before each QRS complex with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Therefore, this is a sinus bradycardia. The QRS complex duration is prolonged (0.12 sec). It has a morphology that resembles a left bundle branch block (LBBB), but there appears to be a small Q wave in aVL (^), which indicates normal septal activation via a septal branch that originates from the left bundle. With a LBBB there is not conduction through the septal branch and hence there is no normal septal activation seen. Hence this is more likely a nonspecific intraventricular conduction delay. The axis is 0° (positive QRS complex in lead I and isoelectric in lead aVF). The QT/QTc intervals are prolonged (520/470 msec) but are normal when accounting for the prolonged QRS interval (500/450 sec). There is low voltage in the limb leads. Importantly, these sinus QRS complexes are similar, but have a slightly different morphology from the echo beats seen in ECG 117A and 117B. Since the echo beats are also the result of normal AV node–His-Purkinje conduction they should be identical in duration, morphology and axis to the sinus beats. However, the echo beats also have similarities to the ventricular complexes. Therefore, the echo beat is likely fusion between the ventricular complex and ventricular activation via the normal AV node–His-Purkinje system. ■

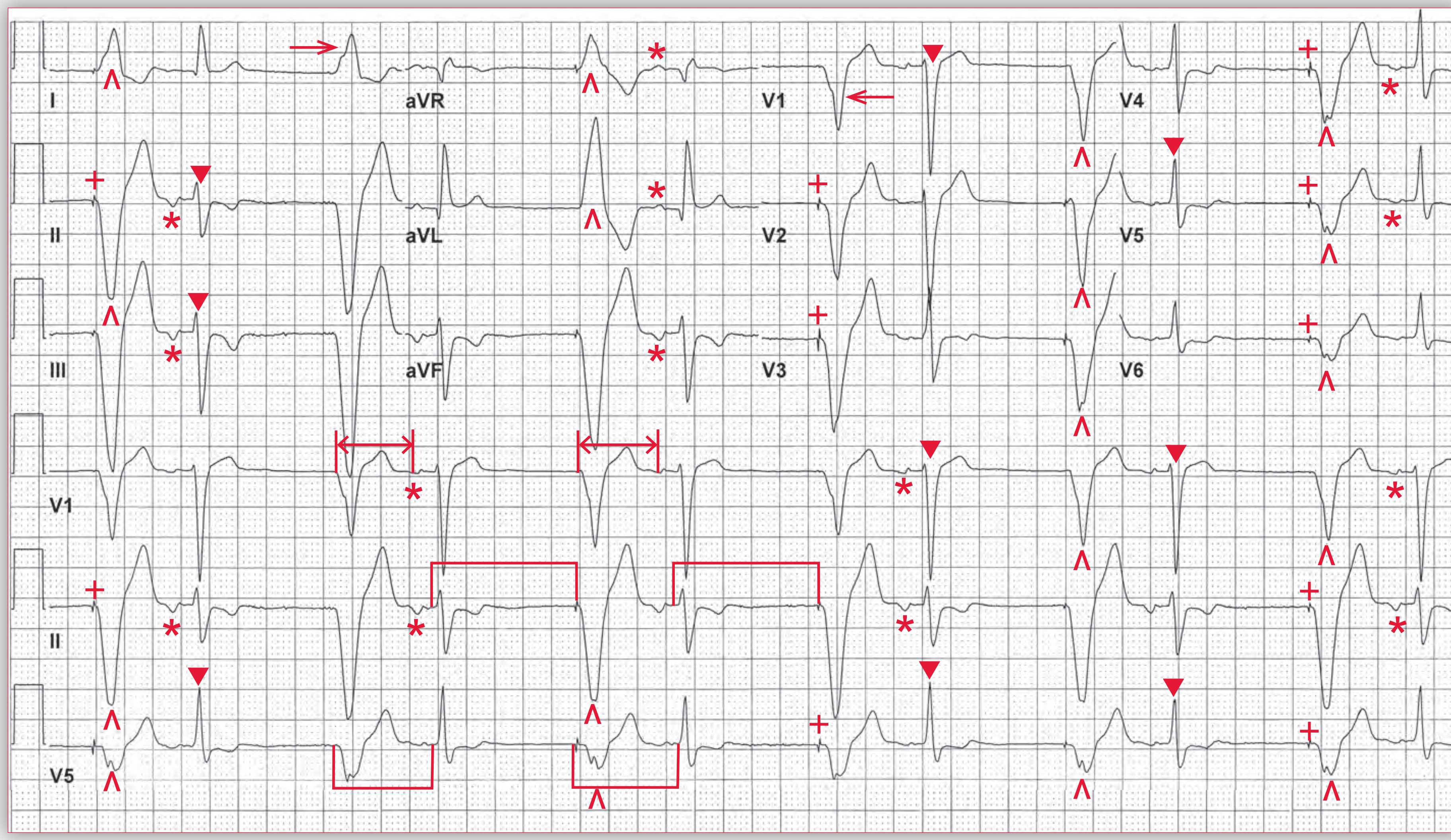
Notes

A 66-year-old woman presents to her doctor's office for a routine check up. She feels well. Her physician notes an irregular heart rate on physical exam and performs an ECG.

What does it show?

Is the pacemaker functioning normally?





ECG 120 Analysis: Ventricular paced rhythm, retrograde (VA) conduction, echo beats

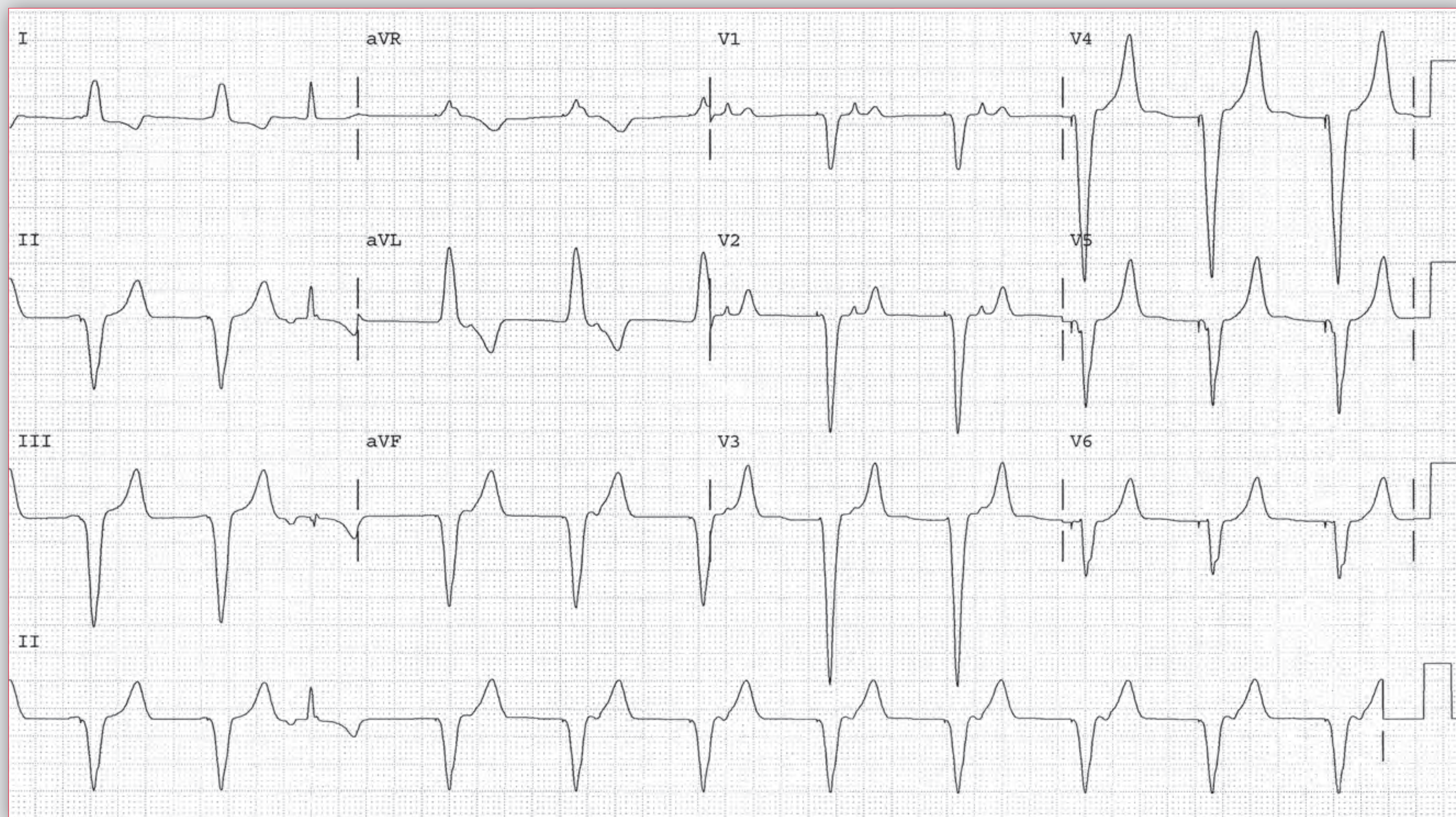
The rhythm is regularly irregular with a repeating pattern of short (⊓) and long (⊔) RR intervals. The average rate is 72 bpm. There are QRS complexes with a prolonged duration (^) (0.18 sec) and a left bundle branch block morphology with a broad R wave (→) in lead I and QS complex in lead V1 (←). A pacemaker stimulus (+) can be seen before each of these complexes; there is no P wave before any of these wide QRS complexes. Hence these are ventricular paced complexes and the pacemaker is functioning in a VVI mode, as there is no evidence for atrial activity or atrial pacing stimulus before these complexes. After the paced complex, there is a negative or retrograde P wave (*) with a fixed RP interval (↔) (0.56 sec). This retrograde P wave is a result of retrograde conduction of the ventricular paced complex and VA (retrograde) conduction through the AV node to activate the atrium. Following the retrograde P wave there is a narrow QRS complex (▼) with a duration of 0.10 sec. The PR interval is constant (0.18 sec). The QRS morphology is normal, and there is a normal axis between 0 and +90 (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/390 msec).

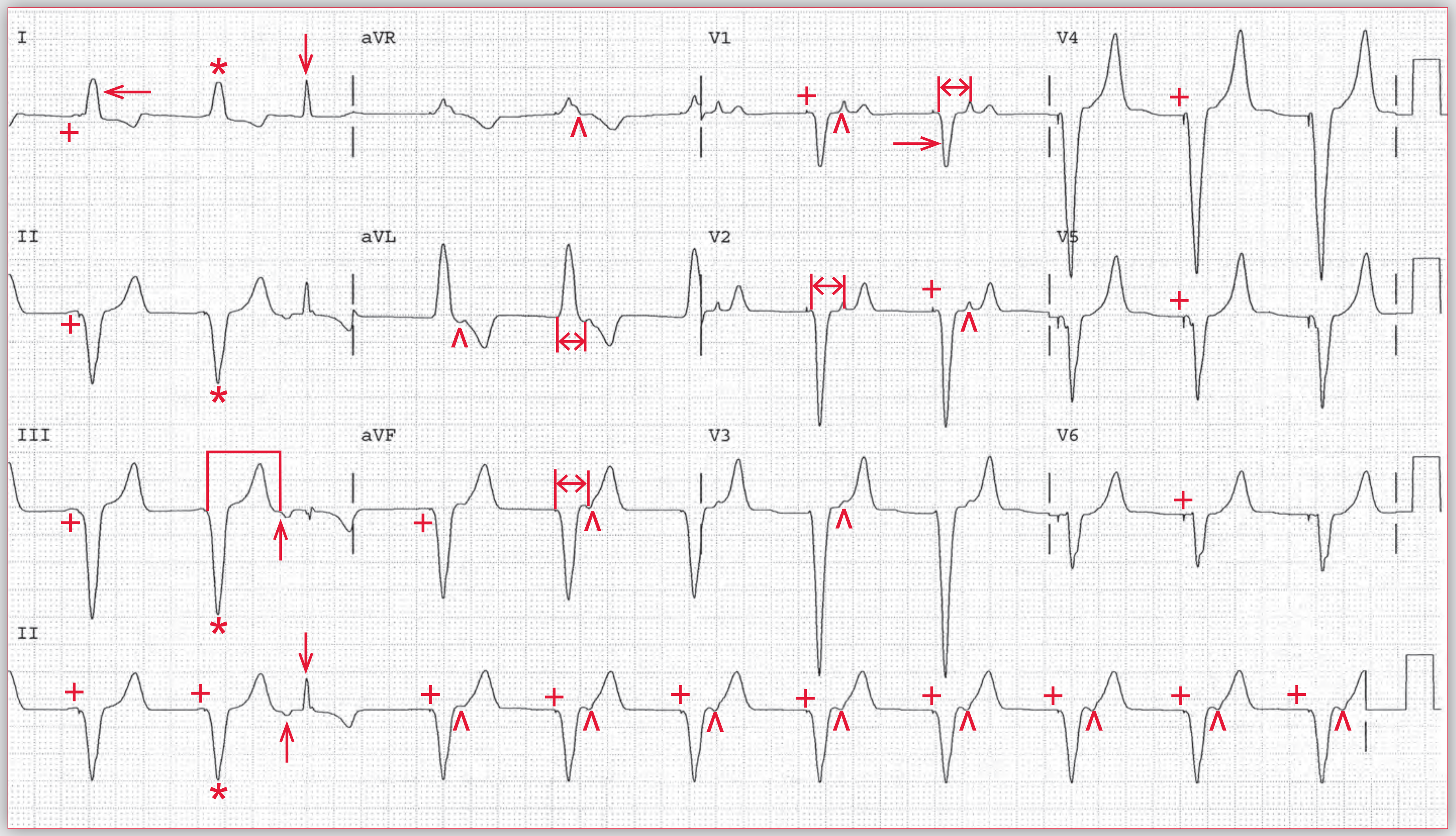
The lower rate limit of the pacemaker is 60 bpm, *ie*, the interval between the narrow complex and the paced QRS complex (⊓). The narrow QRS complex is in response to the retrograde P wave, which is due to VA conduction from the ventricular paced complex. This is termed an echo beat, *ie*, the retrograde atrial impulse results in antegrade conduction through the AV node, and then there is conduction through the His-Purkinje system producing a supraventricular complex. Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes, or junctional complexes. The VA conduction may occur via an accessory pathway (overt or concealed), one of two AV nodal pathways (dual AV nodal pathways), or through the normal AV node. In the latter situation, the retrograde atrial activity is appropriately timed such that the AV node is able to conduct an impulse antegradely. Regardless of how the impulse travels back to the atrium, the antegrade ventricular activation is via the normal AV node–His-Purkinje system as the QRS is narrow and resembles the sinus QRS complex. ■

Notes

A patient is seen in the pacer clinic for routine follow-up and pacemaker check. An ECG shows occasional narrow QRS complexes.

What is the most likely explanation?





ECG 121 Analysis: ventricular paced rhythm with retrograde (VA) conduction and an echo beat

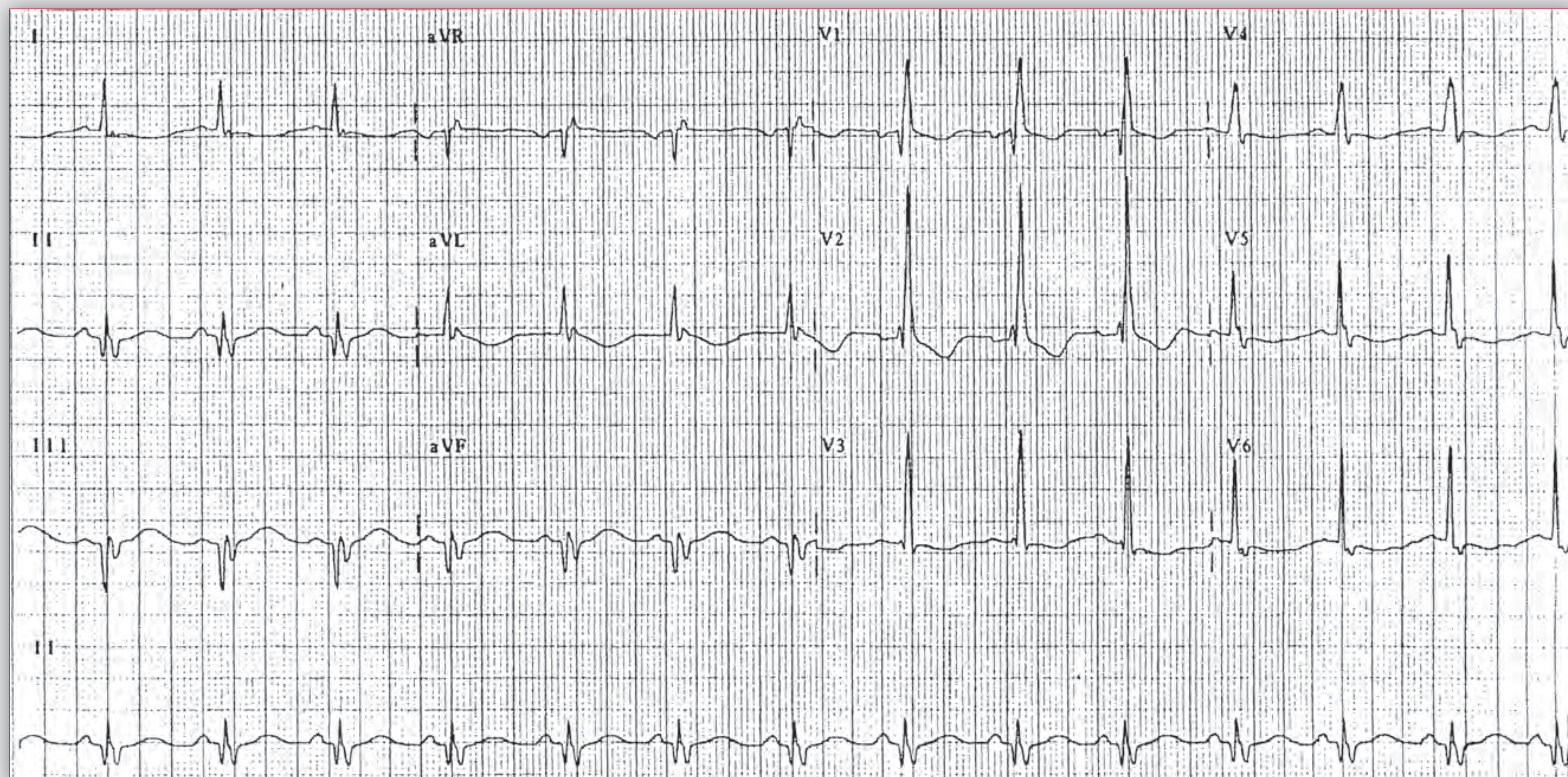
There is a regular rhythm at a rate of 62 bpm. The QRS complex duration is prolonged (0.16 sec), and a small pacemaker stimulus (+) can be seen before each QRS complex. Hence these are ventricular paced complexes. There are no P waves seen before any of these paced QRS complexes. The QRS complex in lead I has a broad R wave (←) and there is a QS complex in lead V1 (→). Hence this is a right ventricular pacemaker functioning in a VVI mode. The QT/QTc intervals are prolonged (500/510 msec) but are normal when the prolonged QRS complex is considered (440/450 msec).

There is a negative waveform (^) seen after the QRS complexes (*ie*, complexes 4–11) that are retrograde P waves. They have a fixed RP interval (↔) (0.24 sec). There is a negative waveform (↑) seen after the second paced QRS complex (*). Although this could represent a premature atrial impulse, the fact that there is evidence for retrograde atrial activity with other QRS complexes makes it more likely that this is also a retrograde P wave with a longer RP interval (⊓) (0.60 sec) than the other retrograde P waves. Following this P wave is an early and narrow QRS complex (↓) (duration of 0.08 sec); the PR interval is 0.16 sec. This complex, which is responding to the P wave before it, results from retrograde or VA conduction to the atrium with subsequent atrial activation and then antegrade conduction of the impulse via the normal His-Purkinje system. This is an echo beat. Echo beats can occur whenever the preceding QRS complex is not preceded by a P wave and hence is associated with VA (retrograde) conduction to activate the atrium. This occurs with paced complexes, ventricular complexes, or junctional complexes. In this case, there is a critical and longer VA conduction time necessary for the echo beat to occur, *ie*, the RP interval is longer when compared to the RP interval of the other paced complexes (*ie*, 0.60 vs. 0.24 sec). There are two potential explanations for the longer VA conduction time. It may be the result of autonomic inputs that slowed retrograde conduction. Alternatively, the longer VA conduction time is the result of retrograde conduction via a slow AV nodal pathway with antegrade conduction back to the ventricle via a fast AV nodal pathway. In contrast, the other paced complexes are associated with VA conduction via the fast pathway and hence the appropriate conditions for an echo beat are not present. ■

Core Case 122

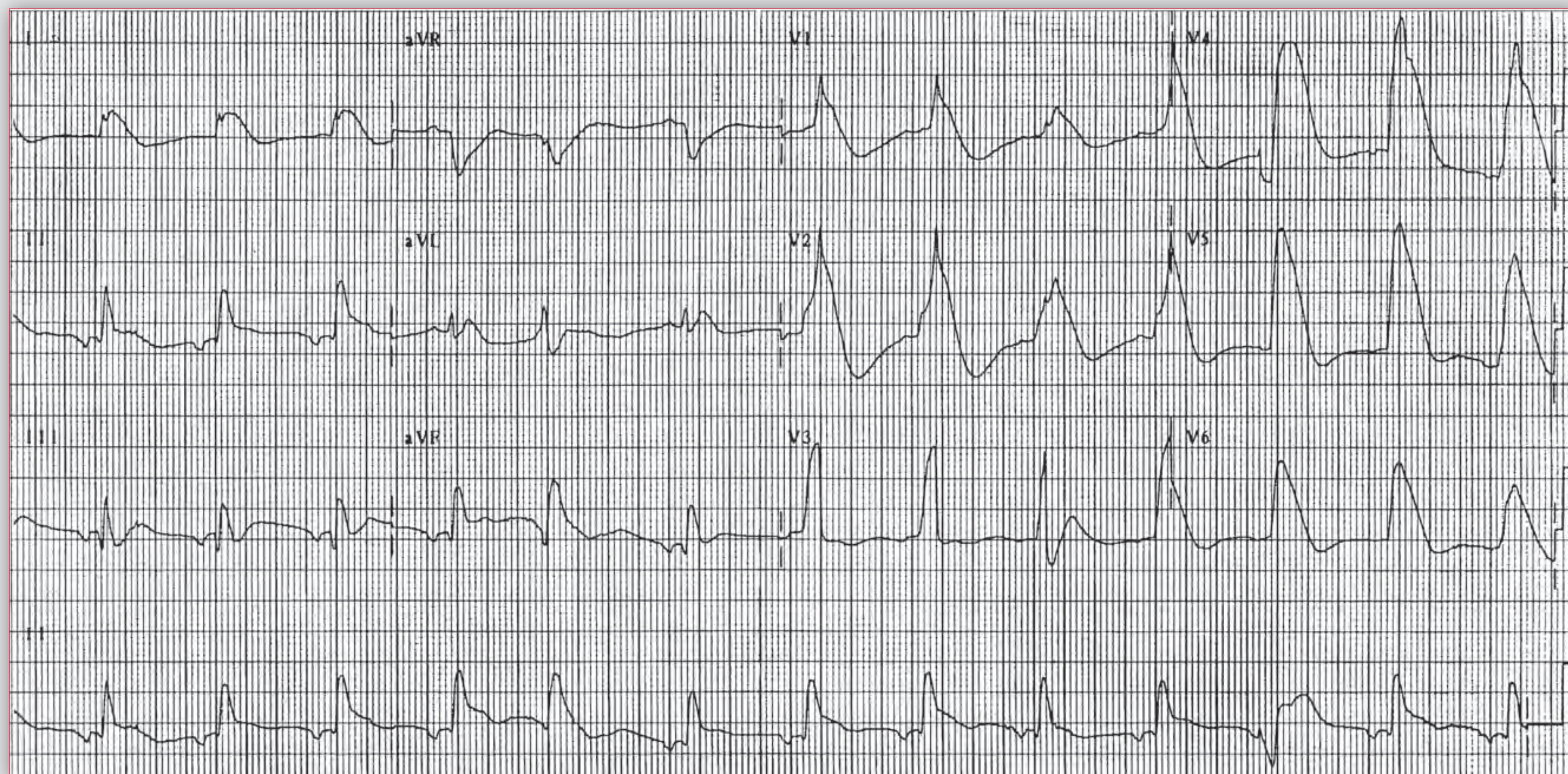
A 68-year-old man with a history of previous myocardial infarction is seen for a routine examination. The baseline ECG is shown (ECG 122A). One week later, he develops atrial fibrillation and undergoes electrical

ECG 122A



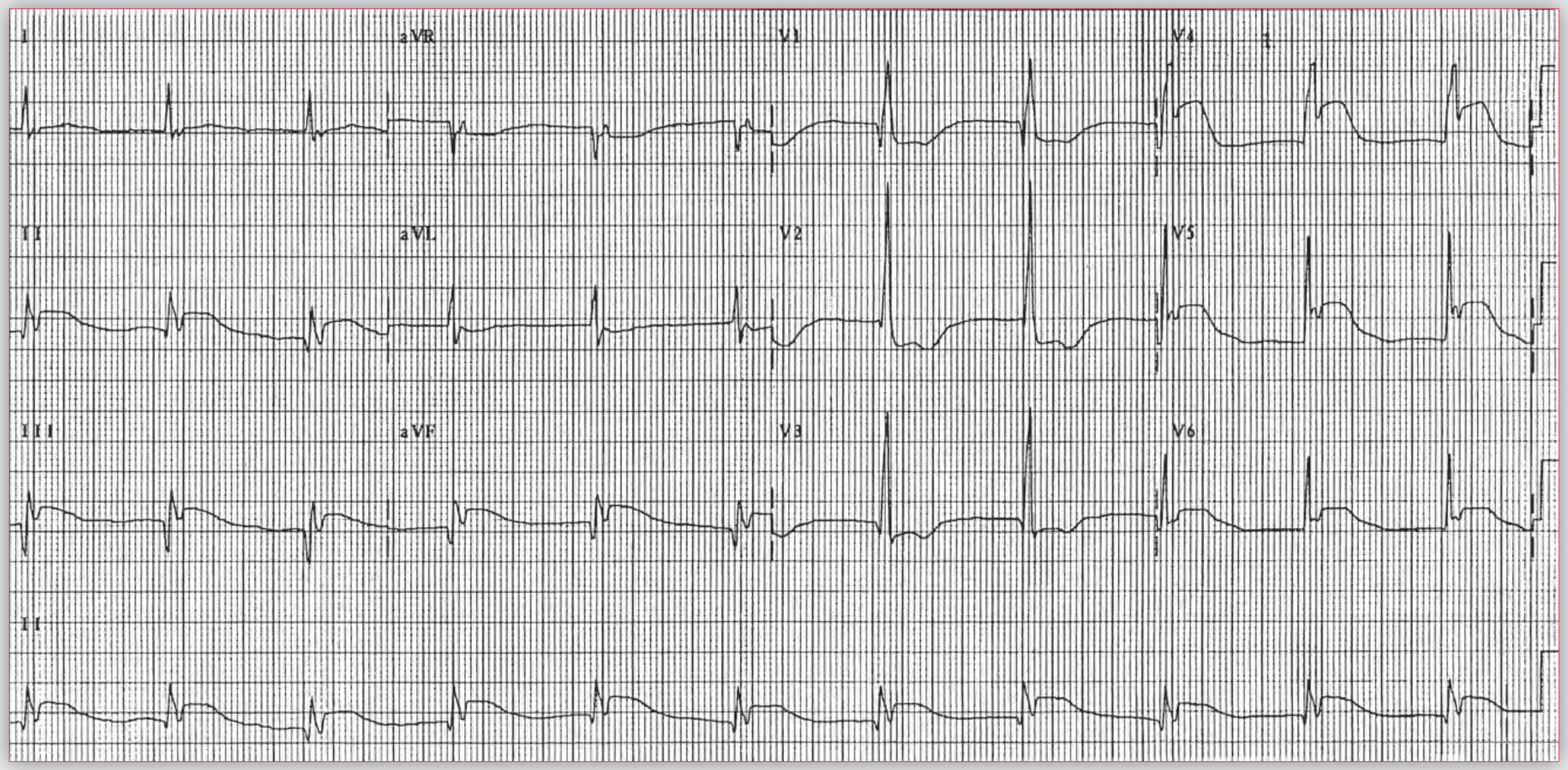
cardioversion for it. Immediately after cardioversion, another ECG is obtained (ECG 122B), and the cardiologist is concerned. An ECG is repeated 5 minutes later (ECG 122C).

ECG 122B



Core Case 122

ECG 122C

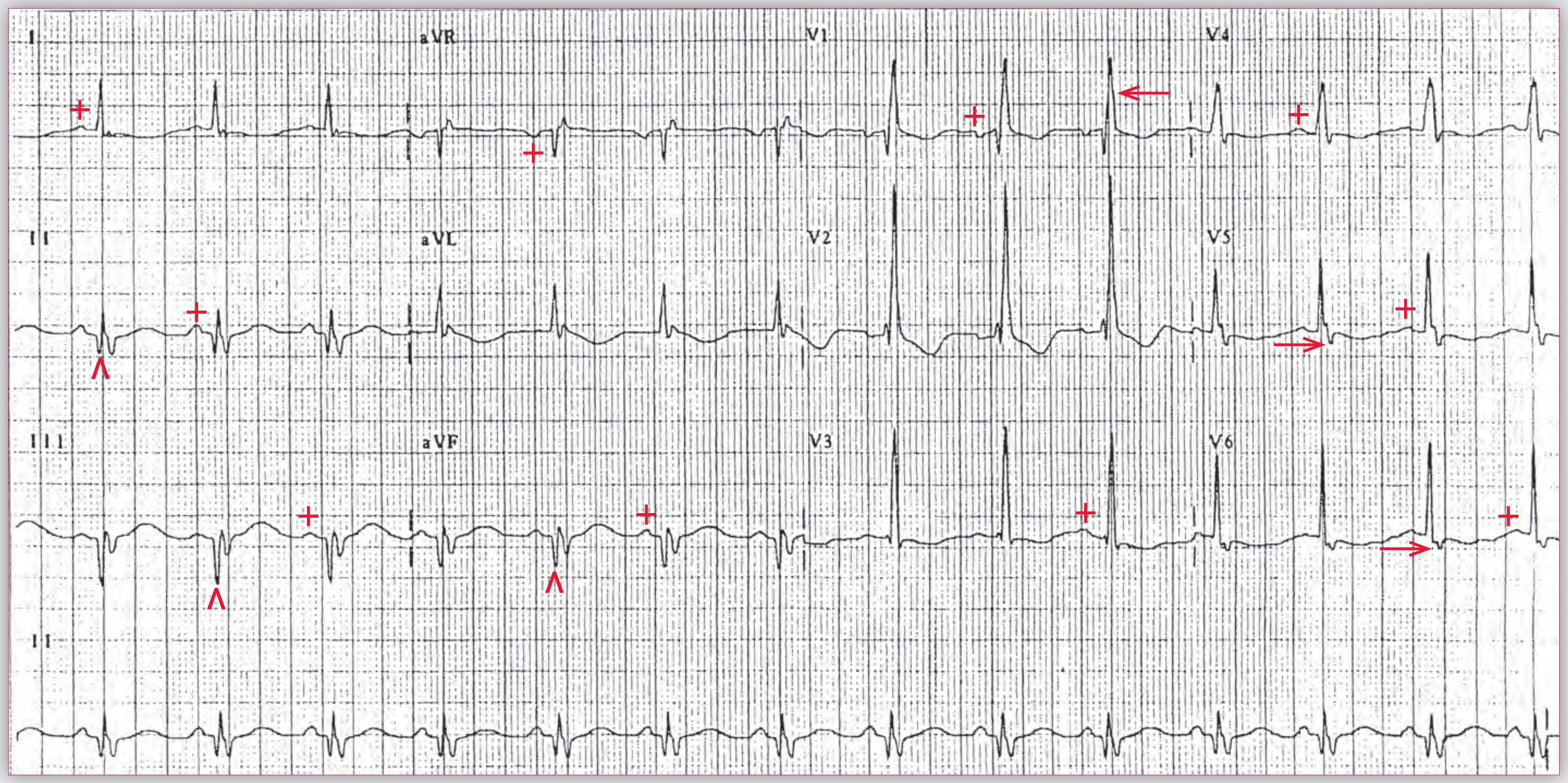


Core Case 122

What abnormality after cardioversion is the reason for concern?

What is the etiology of this abnormality?

Is further therapy necessary?



ECG 122A Analysis: **Sinus rhythm, prior inferior wall myocardial infarction, right bundle branch block**

ECG 122A shows there is a regular rhythm at a rate of 96 bpm. There is a P wave (**+**) before each QRS complex with a stable PR interval (0.16 sec), and the P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm. The QRS complex duration is prolonged (0.14 sec), and there is a pattern of a right bundle branch block morphology with a broad R′ in lead V1 (←) and a terminal S wave in leads V5–V6 (→). There is a normal axis between 0° and +90° (QRS positive in leads I and aVF). There are Q waves (^) in leads II, III, and aVF, indicative of an old inferior wall myocardial infarction. The QT/QTc intervals are prolonged (400/505 msec) but are normal when the prolonged QRS complex duration is considered (360/455 msec).

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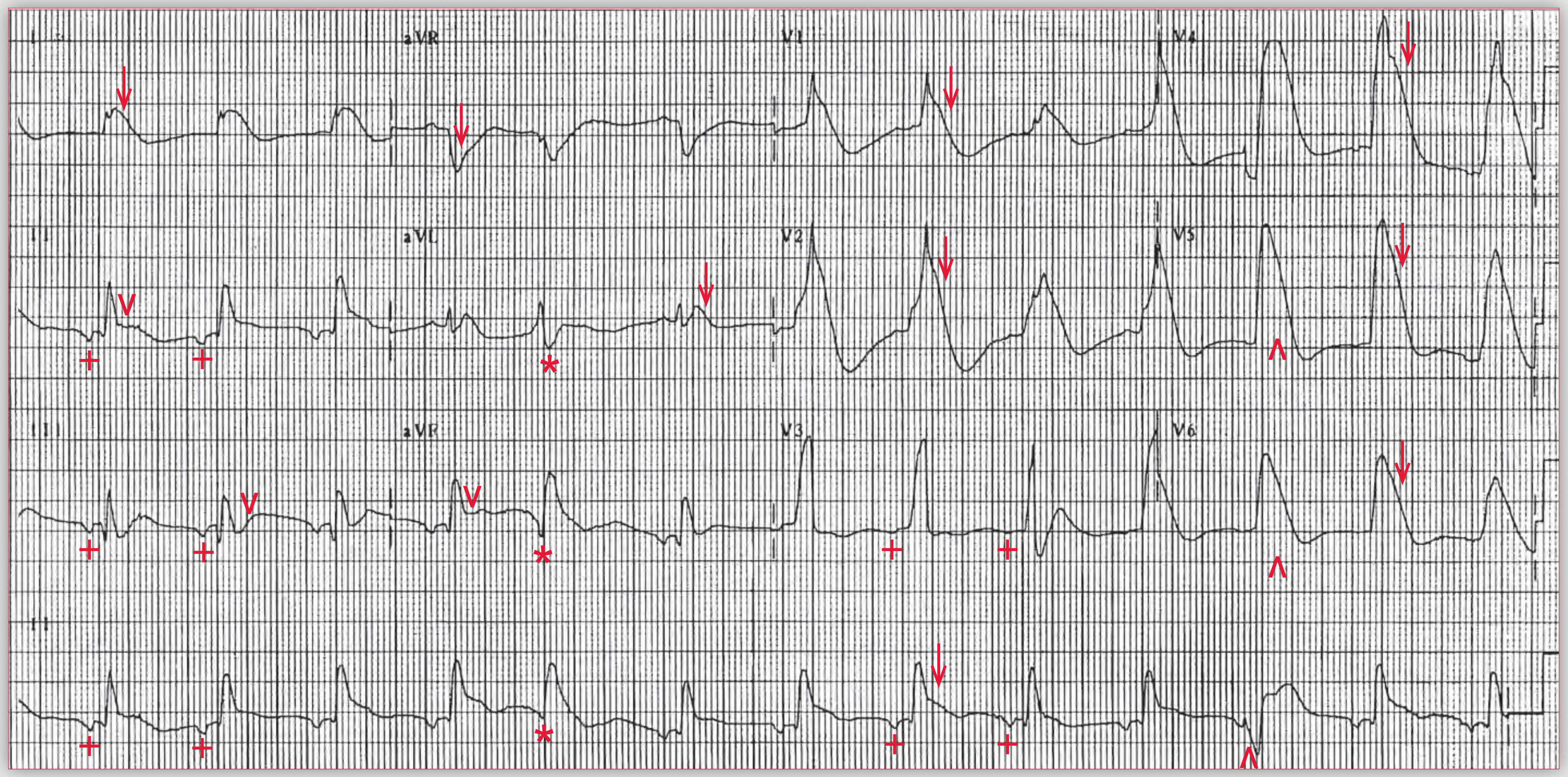

ECG 122B Analysis: Ectopic atrial rhythm, ST-segment elevations due to "current of injury" after cardioversion

ECG 122B shows the patient developed atrial fibrillation and underwent cardioversion. This is the initial ECG obtained immediately after the cardioversion. There are P waves seen (+) that are negative in leads II, III, and aVF. Hence this is an atrial rhythm. The fifth QRS complex is premature (*), and it has the same morphology as the other QRS complexes. No P wave is seen before this complex and hence it is probably a premature junctional complex. The eleventh QRS complex (^) is also premature. It is wider than the supraventricular complexes and has a different morphology. This is likely a premature ventricular complex.

Noted are dramatic ST-segment elevations (↓), especially in the precordial leads as well as leads I and aVL. There is also minor ST elevation in leads II, III, and aVF (v). The QRS complexes have a morphology that resembles an acute anterior wall myocardial infarction; this is known as a current of injury. ST elevation resembling an acute myocardial infarction or a current of injury may be seen after cardioversion or defibrillation, although this is not common. These acute changes are not indicative of a myocardial infarction and often resolve within 2–5 minutes after the delivery of an electric shock.

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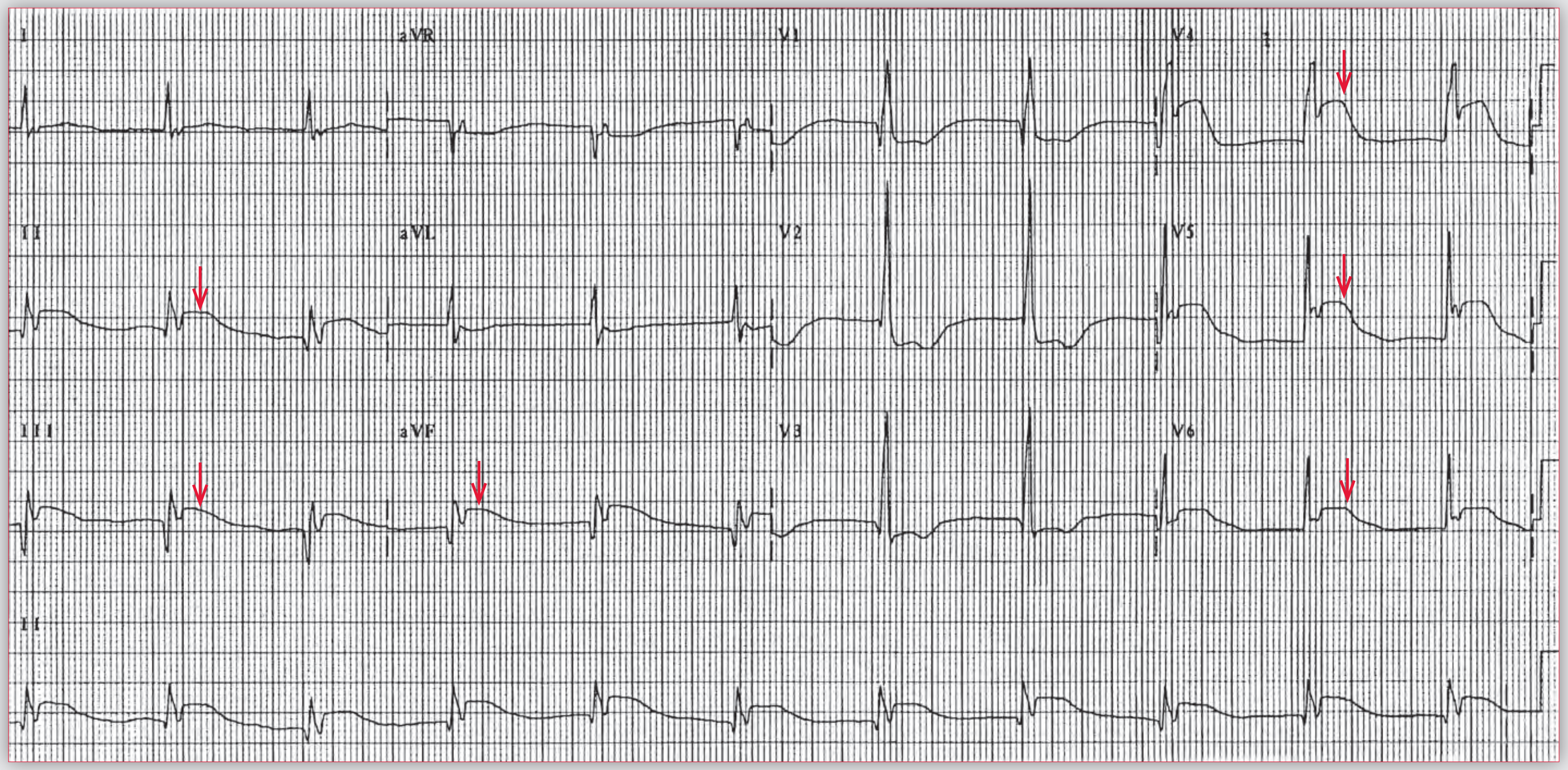


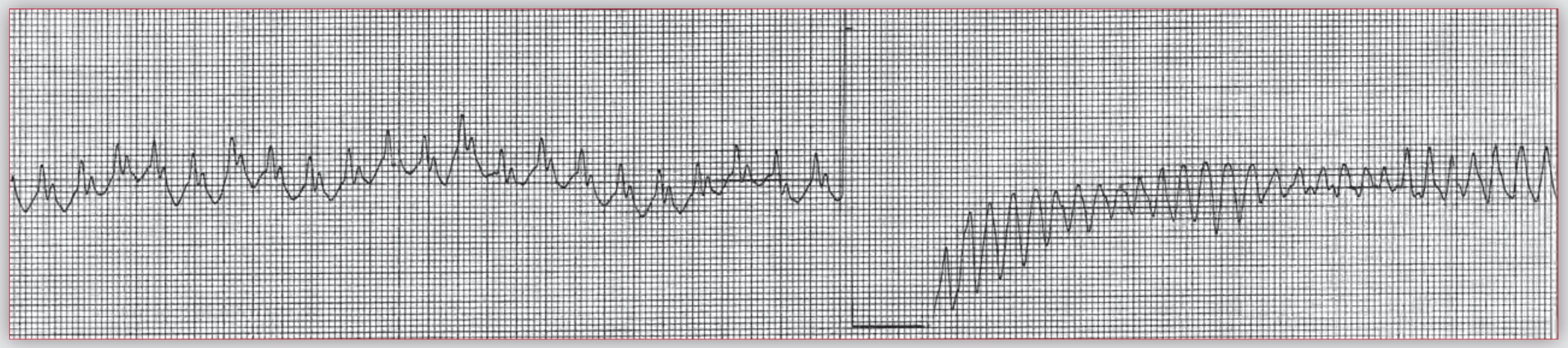
ECG 122C Analysis: Resolving current of injury

Five minutes after cardioversion, **ECG 122C** was obtained. Although ST-segment elevations (↓) are still present, they are less dramatic and are resolving. ■

Core Case 123

Case 123A: A 56-year-old man is admitted to the hospital with chest discomfort. He is placed on telemetry and treated with intravenous nitroglycerin and heparin. Two hours after admission, he is noted to have a rapid, wide complex tachycardia and no blood pressure is obtained. CPR is initiated and an electric shock from a defibrillator is given (ECG 123A). There is a change in the rhythm but a blood pressure is still not obtained.

ECG 123A

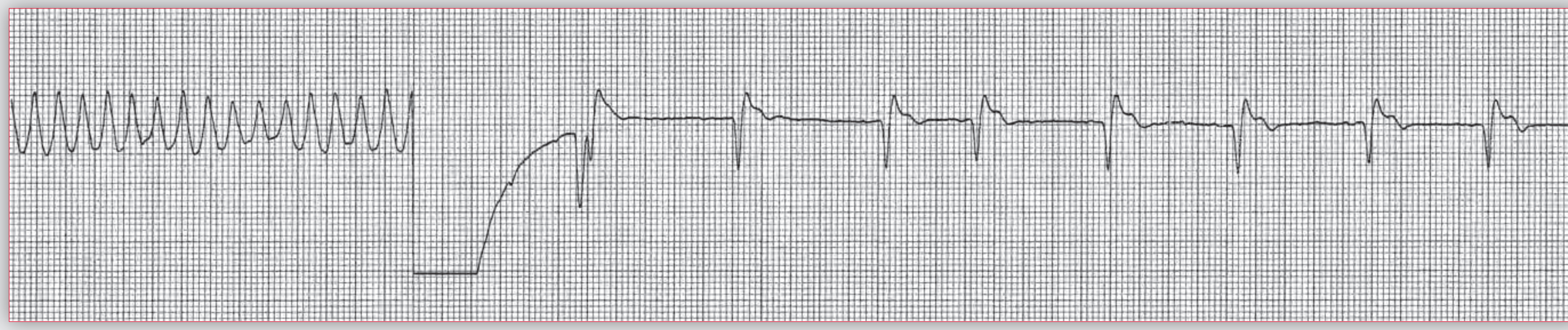


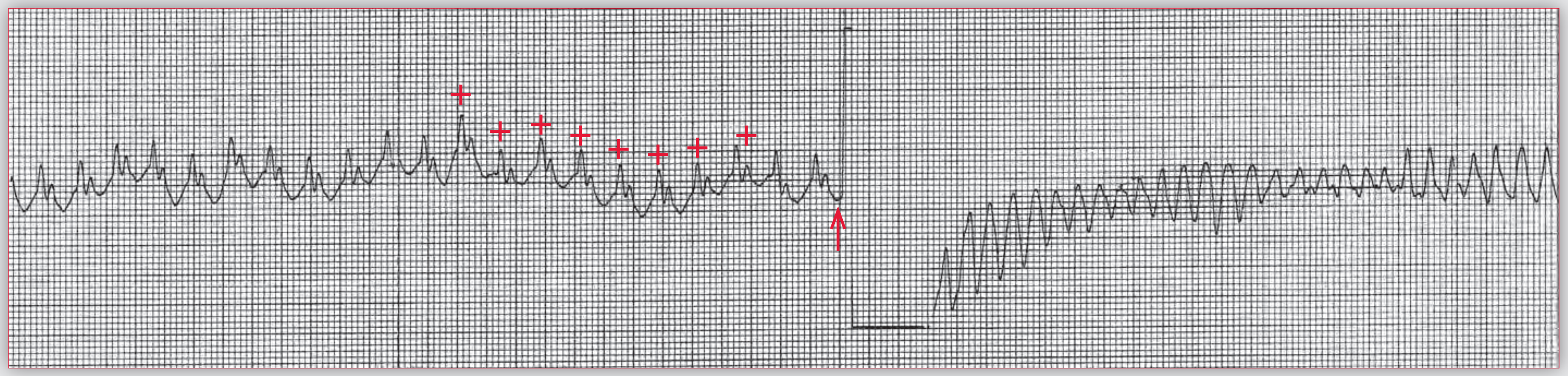
Case 123B: A 48-year-old woman with a known dilated cardiomyopathy is admitted to the hospital for decompensated heart failure. She is placed on telemetry and begun on an intravenous infusion of furosemide. Several hours after admission, a wide complex tachycardia is noted on telemetry. No pulse is obtained and CPR is begun. An electric shock is delivered, after which a slow, narrow complex rhythm is noted. A pulse is now obtained.

Why was the electric shock in Case 123A ineffective?

What accounts for the success of the shock in Case 123B?

ECG 123B





ECG 123A Analysis: Ventricular tachycardia, low-energy defibrillation (nonsynchronized) shock, with R on T, ventricular fibrillation

In **ECG 123A**, the rhythm strip shows a wide complex tachycardia at a rate of 240 bpm. There are no obvious P waves noted. This is a rapid ventricular tachycardia. It can be seen that the cardioverter/defibrillator delivered a nonsynchronized shock. The energy used was only 50 joules. The electric shock was delivered on the T wave (↑) and resulted in ventricular fibrillation. This is the same as the R-on-T phenomenon.

The use of an electric shock to treat arrhythmias is either defibrillation or cardioversion. Defibrillation is the nonsynchronized delivery of energy, *ie*, the shock is not related to the QRS and is delivered as soon as the button on the machine is pushed. Hence the discharge may occur at any time during the cardiac cycle. In general, high energy levels are required with defibrillation to make certain that an adequate amount of energy reaches the heart so that the entire myocardium is depolarized simultaneously. Often the maximum output of the machine is used, *ie*, 360 joules with a monophasic waveform and 200 joules with a biphasic waveform. Cardioversion is the synchronized delivery of energy, *ie*, the shock is coupled to the cardiac cycle and is delivered simultaneously with the R wave; hence the shock is delivered when the myocardium is already depolarized and hence less energy is required to insure that all of the myocardium is depolarized, especially the reentrant circuit responsible for the arrhythmia.

Defibrillation (with high energy) is the preferred method for delivering an electric shock to depolarize the ventricular myocardium when distinct QRS complex are not present (as with ventricular fibrillation) or when there is no distinct R wave and the QRS complex cannot be distinguished from the T wave. If cardioversion is used in this situation, there is an equal chance for the electrical discharge to be delivered on the R wave or the T wave. When a distinct R wave can be identified, cardioversion should be used, as the termination of an arrhythmia occurs with lower energy levels.

When the machine is set to defibrillation, a nonsynchronized shock is delivered, *ie*, the shock occurs as soon as the button on the machine is pushed. Hence the shock may be delivered at any time during the cardiac cycle. If there is a distinct QRS complex and T wave, there is the possibility of delivering a shock on the T wave that has a potential to provoke ventricular fibrillation if a low energy shock is used. The T wave corresponds to the vulnerable period of the action potential and is a time when the membrane potential is more negative than the resting membrane potential (*ie*, less negative than –90 mv). This is the time during the action potential that the ventricular fibrillation threshold is determined. Generally, a relatively high energy level is necessary to provoke ventricular fibrillation. However, with ischemia the threshold is lower, and even a low energy impulse can provoke ventricular fibrillation. Therefore, in this case the use of low-energy defibrillation (50 joules) delivered the shock on the T wave, resulting in ventricular fibrillation.

In this patient, distinct QRS complexes are seen (+) and cardioversion (delivery of energy synchronous with the QRS complex) should have been used. Since defibrillation was used, a higher energy level would have been more appropriate.

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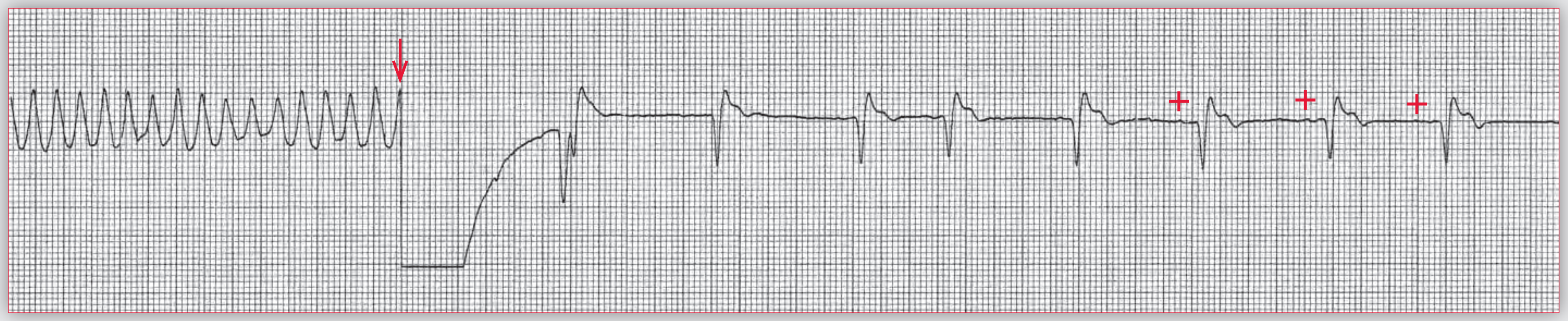


ECG 123B Analysis: Ventricular tachycardia, low-energy synchronized cardioversion, narrow complex rhythm

In **ECG 123B**, this rhythm strip shows a ventricular tachycardia at a rate of 300 bpm. This is often termed ventricular flutter. In this situation, there is a distinct QRS complex seen and the rhythm was appropriately cardioverted using a shock that was delivered synchronous with the QRS complex (↓). Immediately following the shock, there is a supraventricular rhythm with P waves before each QRS complex (**+**). ■

Core Case 124

Case 124A: A 68-year-old man with a history of an ICD implanted for a nonischemic cardiomyopathy associated with recurrent episodes of ventricular tachycardia presents to the emergency department with complaints of episodes of dizziness and shortness of breath that have been occurring several times per week. He is admitted and placed on telemetry. He has an episode several hours after admission.

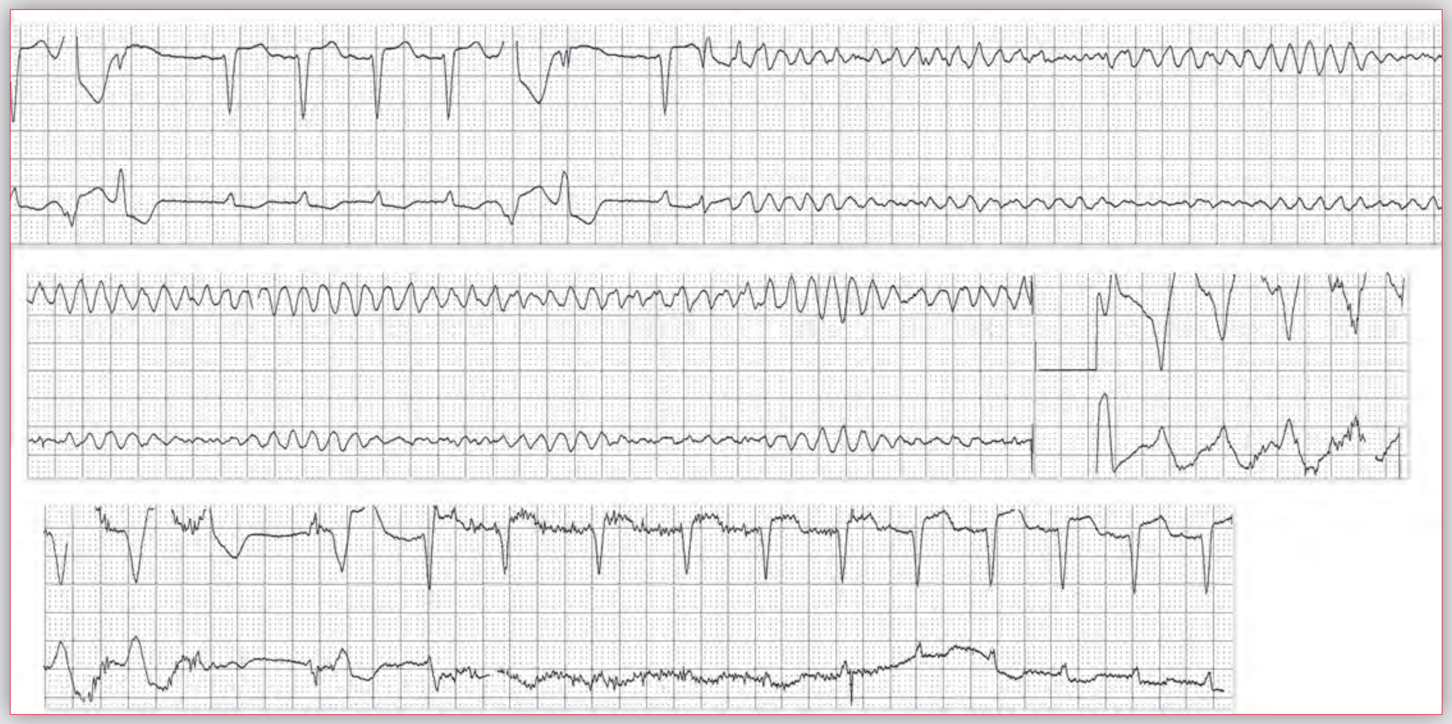
ECG 124A

Case 124B: A 65-year-old man with a history of an ischemic cardiomyopathy presents to the emergency department with a several-hour history of chest discomfort. He states that 2 years before, he had an ICD inserted because of a low ejection fraction (30%), but he has never experienced a shock. His initial ECG does not show any acute changes and initial cardiac biomarkers are negative. He is admitted to the hospital and placed on telemetry. On the following day, he has an episode of chest discomfort and then loses consciousness.

124A: What does the telemetry show?

124B: What is seen on telemetry?

ECG 124B



Core Case 124

Case 124C: A 56-year-old man has a nonischemic cardiomyopathy with an ejection fraction of 25%. He had an ICD placed 6 months prior to being seen in the emergency department for shortness of breath that had become progressively worse over the previous 3 days. In the emergency department, he is noted to have evidence of vascular congestion on chest x-ray and 2+ bilateral peripheral edema. He is treated with intravenous furosemide and admitted for further therapy. He is placed on telemetry. During the night, while sleeping, a rhythm abnormality is noted on telemetry.

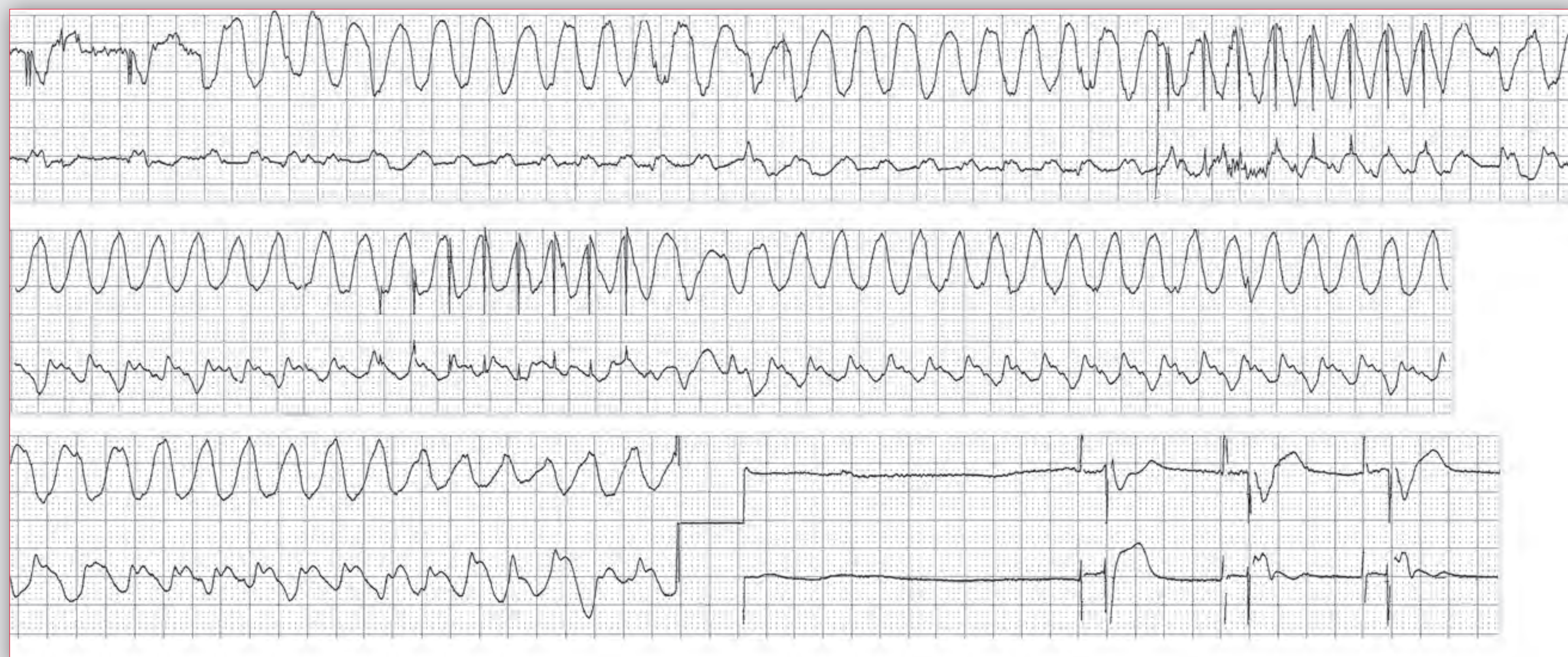
ECG 124C

Case 124D: A 50-year-old woman with a history of a nonischemic cardiomyopathy and an ICD implantation for recurrent ventricular tachycardia is seen in the emergency department because of an ICD discharge that occurred several hours before being seen. Her physical examination is unremarkable. She is admitted to the hospital for observation and placed on telemetry. On the following day she complains of palpitations and then experiences a discharge from the ICD.

124C: What arrhythmia is noted and what is happening with the ICD?

124D: What does telemetry show?

ECG 124D



ECG 124A Analysis: Normal sinus rhythm, ventricular tachycardia, antitachycardia pacing (ATP) with restoration of normal sinus rhythm

ECG 124A is composed of two continuous single channel rhythm strips. The first rhythm strip shows an initial rhythm that is regular at a rate of 90 bpm. There is a P wave (+) before each narrow QRS complex. Hence this is a normal sinus rhythm. The tenth QRS complex (^) is premature and wide with a change in morphology. It is followed by a series of wide complex QRS complexes at a rate of 180 bpm. This is monomorphic ventricular tachycardia. After about 7 sec, there are 8 paced QRS (*) at a rate of 200 bpm (seen on the second strip). This is ATP or burst pacing. This results in restoration of normal sinus rhythm.

Although the ICD will terminate an episode of ventricular tachycardia, it will not prevent this arrhythmia. As this patient is having frequent symptoms that are likely the result of ventricular tachycardia, an attempt should be made to suppress these events. This is not only to treat the symptoms, but also to prevent frequent ICD therapies that will result in more rapid battery depletion.

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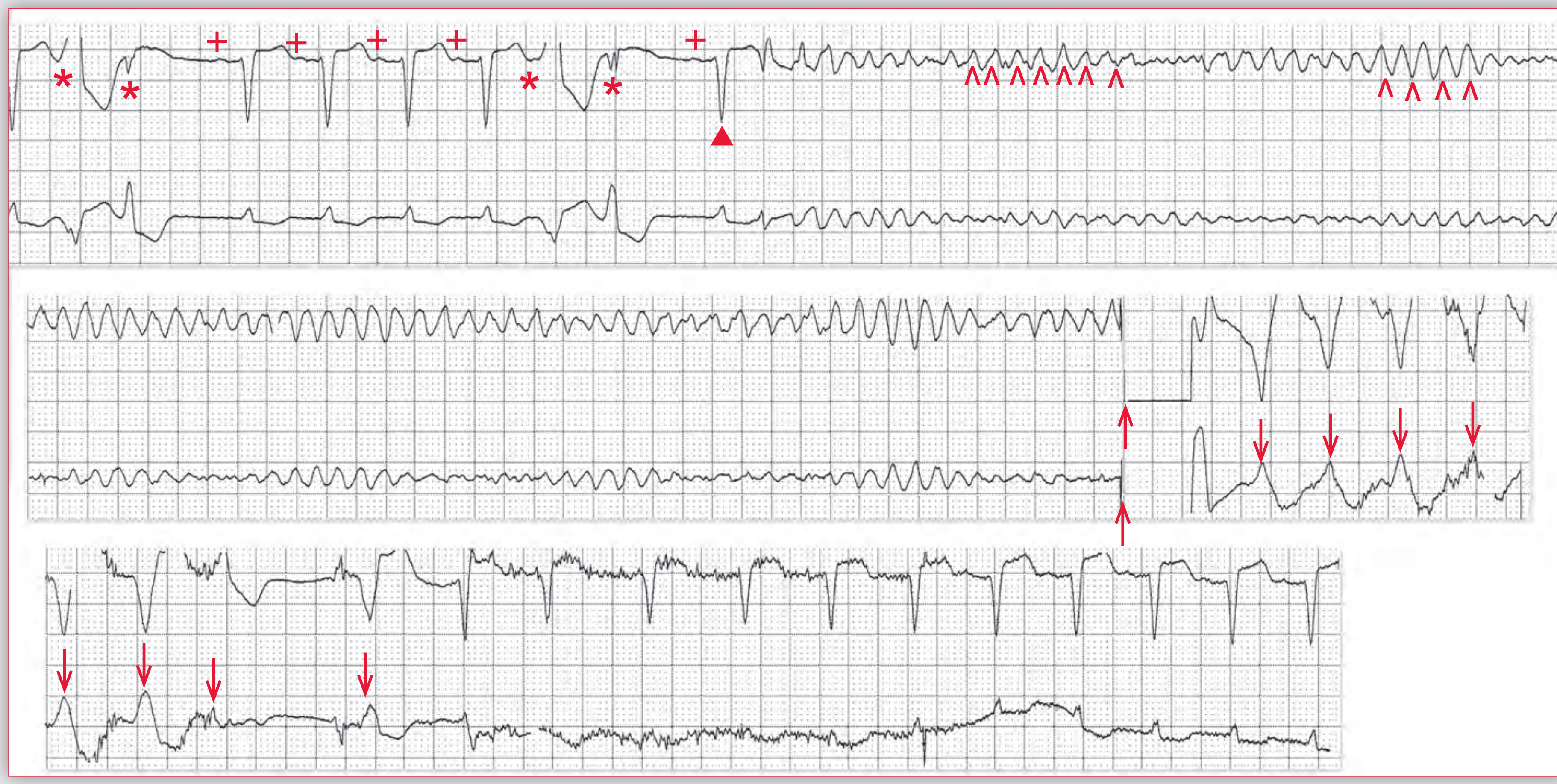

ECG 124B Analysis: Normal sinus rhythm, ventricular fibrillation, restoration of normal sinus rhythm after a defibrillation shock

ECG 124B. These are three continuous dual-channel rhythm strips. The first strip shows an initial rhythm that is regular at a rate of 110 bpm with a P wave (+) before each narrow QRS complex. There are two ventricular couplets noted (*), and after the second ventricular couplet, there is another sinus complex (▲). This is followed by a rhythm that does not have any organized ventricular complexes; in contrast, there are rapid and irregular undulations of the baseline. This ventricular fibrillation. Ventricular fibrillation is seen on the second rhythm strip and after about 13 sec there is a discharge (↑) from the ICD. Following this, there are eight wide complex QRS complexes (↓) at a slow rate, after which there is the resumption of normal sinus rhythm.

Ventricular fibrillation is one of two arrhythmias that is induced by active ischemia, the other being polymorphic ventricular tachycardia. The occurrence of ventricular fibrillation during an episode of chest discomfort in this patient strongly suggests that there is active ischemia that should be aggressively treated.

continues

ECG 124C Analysis: Normal sinus rhythm, ventricular tachycardia, ATP

ECG 124C. There are two single channel rhythm strips seen. The first strip shows 3 narrow QRS complexes that are associated with P waves (+). This is a normal sinus rhythm at a rate of 90 bpm. Following these, there is a wide complex tachycardia at a rate of 180 bpm. This is sustained monomorphic ventricular tachycardia. After several seconds there are eight paced complexes (*) at a rate of 240 bpm. This is ATP or burst pacing. The burst does not terminate the ventricular tachycardia. A second episode of burst pacing (*) can be seen that is also ineffective.

The rate, duration and number of ATP therapies is programmable. However, if several ATP therapies are ineffective, the device is usually programmed to deliver a low- or high-energy discharge for either cardioversion or defibrillation. One potential concern about ATP, especially when a rapid rate is used, is rate acceleration of the ventricular tachycardia that would certainly result in the delivery of a shock. As ATP is ineffective, it might be preferable to deactivate this feature in this patient and rather have the ICD deliver a shock to revert the ventricular tachycardia. This will reduce the rate of battery depletion.

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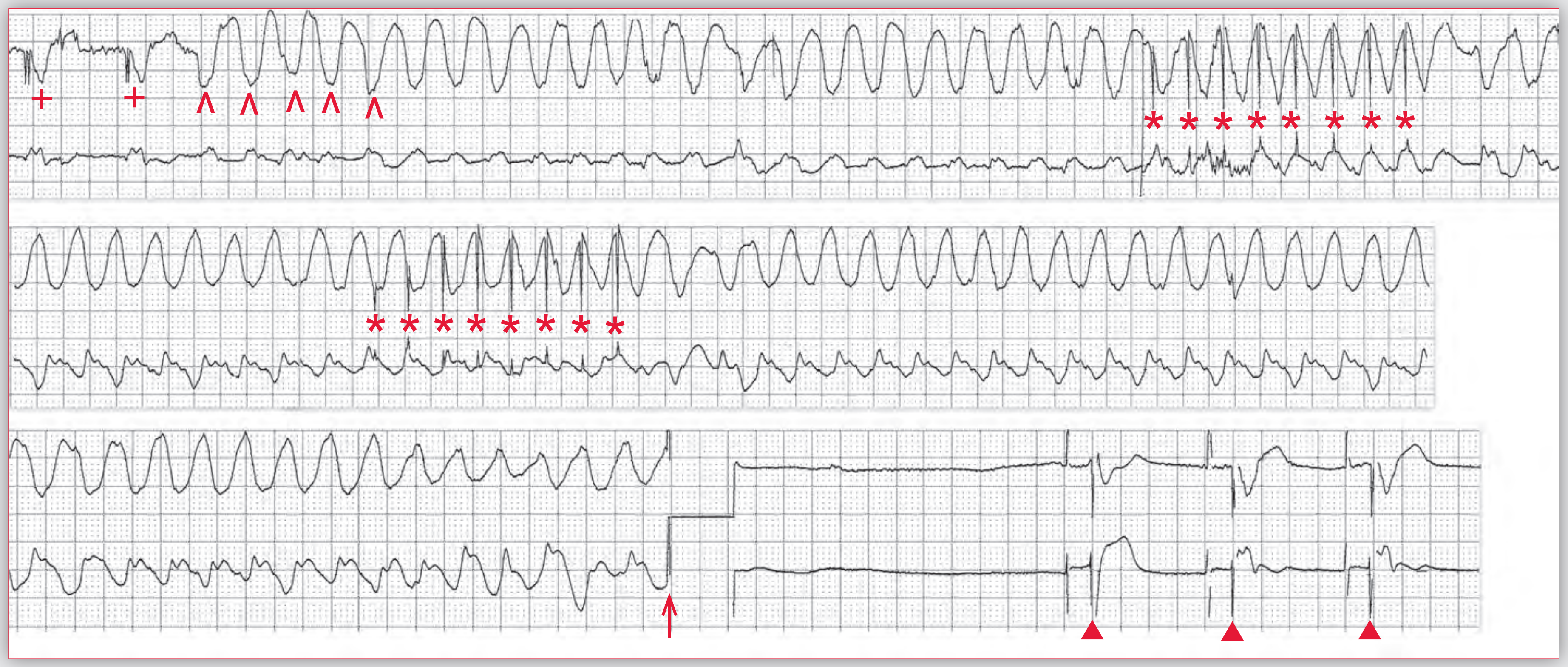

ECG 124D Analysis: AV sequential pacing, ventricular tachycardia, ineffective ATP, low-energy cardioversion shock, restoration of AV sequential pacing

ECG 124D. There are three continuous two-channel rhythm strips. The first strip shows two AV sequential paced complexes (+) after which there is a wide complex tachycardia (^) at a rate of 240 bpm that shows subtle changes in morphology. This is an episode of sustained monomorphic ventricular tachycardia. After several seconds there are 8 beats (*) of a paced rhythm at a rate of 260 bpm. This is ATP or burst pacing that fails to terminate the ventricular tachycardia. Several seconds later, there is a second episode of ATP that is also ineffective for terminating the ventricular tachycardia. Several seconds later, there is a discharge from the ICD (↑) that occurs at the peak of the R wave and results in restoration of AV sequential pacing (▲).

The function of the ICD is dependent upon heart rate. Whenever a certain heart rate is detected (the heart rate that the ICD will detect and respond to is programmable), the ICD will deliver a therapy based on how it is programmed. It may be programmed to deliver ATP, and the rate, duration and number of ATP therapies is programmable. The ICD may be programmed to deliver a low-energy shock (cardioversion) or a high-energy shock (defibrillation).

In general, ICDs can be programmed to provide different therapies to tachyarrhythmias in up to three heart rate zones. This approach is of use since relatively slow ventricular arrhythmias are often tolerated for at least several minutes (*ie*, they are not associated with hemodynamic instability and do not lead to loss of consciousness or other unstable symptoms). They can often be terminated with ATP or low-energy cardioversion that can be delivered quickly. ATP is not usually associated with symptoms, although low-energy cardioversion may cause discomfort. Tachycardias that have more rapid rates are more likely to be unstable and poorly tolerated; they generally require immediate high-energy defibrillation as it can become increasingly difficult to terminate if definitive therapy is delayed.

The ICD can be programmed to deliver sequential therapies, *ie*, up to six attempts of ATP, cardioversion or defibrillation in each rate zone. After each delivered therapy, the device reevaluates the rhythm, and if the tachyarrhythmia persists, the next therapy is delivered. If the arrhythmia persists after the maximum number of attempts for a therapy has been delivered, the device will then deliver the next type of therapy that has been programmed. This has been termed tiered therapy. ■

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